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PERSPECTIVE IN THE EXAMINATION, PROGNOSIS AND TREATMENT IN HEART DISEASE.[1]

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I WAS asked to discuss the examination of a patient with heart disease. No apology is needed when one reflects upon the varying points of view still held concerning such important matters as the relative importance of the heart muscle in the production of congestive failure, or of the electrocardiograph in diagnosis and prognosis. Pardee[(1)] and White[(2)] see prognostic value in the electrocardiogram under certain circumstances. Lewis[(3)] perceives none at all. Lewis always uses X rays to tell the size of a heart. Many others use them rarely in a heart examination. White, although agreeing that the heart muscle was ignored too much in the past, thinks its rôle in connexion with congestive heart failure has become magnified. The subject is therefore one which should give scope for discussion.

Such great changes have occurred in the knowledge of chronic cardiac disease since many of us left the schools that the mental background with which we should approach the investigation of a heart case, has altered radically. That is to say, the sensations arriving in our minds are the same, but our perceptions should be very different. Principles belonging to the background will therefore be reviewed before stock is taken of what clinical methods we have at our disposal.

The Diagnosis-Complex.

Diagnosis of a heart case is of value only in so far as it provides a basis for prognosis and treatment. There may be some cardiac patients in whom

1 Read at a meeting of the Queensland Branch of the British Medical Association on May 5, 1933.

it is just a matter of labelling the case and then treating the label. But in the great majority of cases a single label is not possible, either for the purpose of treatment or for prognosis. Besides the want of infallibility in clinical methods for important cardiac purposes, there are two reasons for complexity. First, many cardiac lesions vary in degree, with, consequently, very varying prognosis and treatment, for example, in valvular disease. Secondly, complex permutations and combinations may occur between the common condition of hypertensive heart disease, another common condition, coronary sclerosis, and the majority of cardiac lesions; and sometimes syphilitic aortitis or myocarditis may be added also, as well as focal sepsis from teeth or tonsils, myxœdema or anæmia *et cetera*. A complicated diagnosis, therefore, may be necessary in any suspected heart case in order to supply us with a proper prognosis and treatment. A *prima facie* "heart case" may be suspected if a patient complains of pain or distress near the chest, shortness of breath, palpitation or swollen ankles, and sometimes also of giddiness, fainting, fatigue, indigestion or hæmoptysis. The investigation should then be such as to supply a diagnosis-complex compiled from the following data:

(1) Ætiological (as given later).

(2) Pathological: Valves, junctional tissues, ventricular myocardium, walls, arteries, (aorta), pericardium.

(3) Functional: (i) No shortness of breath, palpitation or precordial distress with ordinary effort; (ii) shortness of breath, palpitation or precordial distress on hurrying fast or on hills; (iii) cardiac symptoms with ordinary walking or on a short flight of steps (signs of congestion may be present); (iv) cardiac symptoms while at rest (congestion may be marked).

Any "heart diagnosis" should include one or more items out of each of the first two groups and one item (including signs of congestive failure, if present) from the third group. ("Walls" is to remind one to include the size of the heart.) For example, in a case of mitral stenosis other pathological disorders may be present, such as auricular fibrillation or chronic mediastino-pericardial adhesions. The ætiological factor also must be sought for. The original cause may have been rheumatic fever, but septic tonsils might be poisoning the constitution still. The functional efficiency is most important. If the patient be pregnant, she ought to be able to carry on to term, if conforming to (3) (i), but not if to (3) (iv), whilst abortion will probably be necessary with (3) (iii) also, and the question will have to be seriously considered at times with (3) (ii). All this may seem commonplace, but how many of us can say we have not at some time missed an important underlying factor by being satisfied with the diagnosis of auricular fibrillation? One should not be content with the fact that auricular fibrillation is present. It is necessary to find the cause. If the patient can be proved to be suffering from hyperthyreoidism, the treatment is far more than giving digitalis, namely, iodine and removal of three-quarters of the gland. It is only by keeping a scheme like this in the back of our minds that we can avoid some of our mistakes.

CASE I.—I was called to see a patient with auricular fibrillation. The medical practitioner in charge told me there was nothing further to do, as she was now on maintenance doses of digitalis. He added, however, that she required the equivalent of 0·18 gramme (three grains) of standardized digitalis leaf *per diem* to keep her pulse down. When I first saw the patient, her main complaint was nausea and loss of appetite for a week. The gastric symptoms stopped on stopping the digitalis. Her original main complaint had been fatigue, and this was due largely to severe alveolar sepsis. The removal of the dental poisoning was a more important therapeutic necessity than blocking the auriculo-ventricular node with another poison. The patient is again taking a maintenance dose of digitalis leaf, but now only 0·09 gramme (one and a half grains) a day.

Clinical Principles.

The above scheme for a diagnosis, or rather, diagnosis-complex, is but the aim of the investigation. The principles in the mental background consist of more than this. Four fundamental points of view and one subsidiary point of view should underlie a mental approach to the investigation of any given heart case. These are: (*a*) ætiological factors, (*b*) the ubiquity of the nervous system, (*c*) the paramount importance of the muscle which directly fulfils the function of the heart—the pumping muscle of the ventricles (which I will call the "heart muscle"), (*d*) the heart muscle paradox, the heart muscle having such wonderful power of recuperation that ill health in chronic heart disease is primarily due, as a rule, to disease in another structure of the heart. The fifth viewpoint, (*e*), is the twofold course in the very common ætiological factor, coronary sclerosis. The heart muscle may become more or less suddenly overwhelmed, or, more accurately, a part of the heart muscle may become more or less suddenly strangled, in one group, but not in the other, so that in the former there is grave danger of either sudden death or serious crippling.

Ætiology in Heart Disease.

Either the prognosis or treatment or both depend in a very large degree on the ætiology. In the perspective of a cardiac investigation ætiology therefore occupies a prominent position, and each factor should be kept in mind in the examination of any heart case. For example, even when one is satisfied that post-rheumatic or arteriosclerotic changes are actually operative, some attempt should be made to exclude syphilis, though it be sometimes, perhaps, only by inquiry of the past and seeing if there be any signs, such as healed leg ulcers. The first four of the following factors are the most common primary causes of heart disease. Of these essential hypertension is far and away the commonest. Arteriosclerosis is a separate condition. Arteriosclerosis does not cause hypertension. But the development of coronary sclerosis is aggravated by hypertension.[7] Furthermore, coronary sclerosis is the important underlying factor in assisting in the production of myocardial failure in hypertensive

heart disease, whether the failure be sudden or gradual.[7] There are seven other groups which either, on the one hand, tend to act rather as auxiliary causes to other ætiological factors already operating or, on the other hand, are less common. For example, focal sepsis is rarely a primary cause of organic heart disease, but, when a primary cause is operating already, it is a common ancillary cause of myocardial weakness, at any rate in this generation. Again, influenza has little effect on the normal heart, but it is a common precipitating cause of congestive failure in hypertensive disease.

A rare instance of heart disease apparently does occur as a result of focal sepsis.

CASE II.—A well developed young married woman of twenty-six had many stumps, left for years behind dentures, the roots capped by sepsis and the gums very angry with pyorrhœa. It is now two years since she developed puerperal fever after the birth of her third child. In the course of this she suffered from successive thromboses of the large veins in one leg and both arms, and from congestive failure and orthopnœa for many weeks. In the middle of this period (when I saw her first) she complained of severe constricting epigastric pain for several hours. She stated that the pain resembled that of the third stage of labour. The electrocardiograph, which, however, had not been used before, on the next day revealed a pointed inverted *T* wave in Leads I and II, probably not due to digitalis. Anyhow, typical loud gallop rhythm was heard at the apical region for two days five days later. She was most desperately ill, and the temperature did not keep near the normal line until nearly two months after the onset, but she recovered. The mouth was gradually cleared up later. She is well today, and the electrocardiogram is normal, the only obvious reminder of her illness being large veins across the front of her chest. What the underlying cardiac pathology was I do not know, but, whatever it was, the most unusually foul condition of the mouth was most probably responsible. No evidence of arteriosclerosis can be detected, and I am of the opinion the prognosis is good.

The ætiology in heart disease may then be set out as follows:

Four common primary groups:

(1) "Rheumatic infection", frequent sore throats *et cetera*:
  (a) Active.
  (b) Past.
(2) Hypertensive disease.
(3) Coronary sclerosis.
(4) Syphilis.

Seven either ancillary or less common groups:

(5) Chronic focal sepsis.
(6) Deficiency states, for example, myxœdema, Addison's anæmia, hypoinsulinism, hypoglycæmia, secondary anæmia, obesity, unfitness, beri-beri.
(7) Infections other than rheumatic or syphilitic:
  (a) Cardiac organic infectious states, for example, subacute endocarditis.
  (b) General infections, for example, influenza.
(8) Toxic cardiac states, for example, thyreotoxicosis, (increased adrenal action).
(9) Pulmonary heart.
(10) Congenital heart disease.
(11) Trauma:
  (a) Physical injury.
  (b) "Strain" causing "physiological injury" [Fenton *v.* Thorley & Co. (1903) 72 L.J.K.B. 787; (1903) A.C. 443].[m]

There are two other ætiological factors to be considered, but neither causes heart disease. On the contrary, they are causes of nervous disorder.

Ubiquity of the Nervous System.

One of the most difficult clinical problems sometimes is whether a symptom be due to organic heart disease or nervous disorder. The latter may either mimic or complicate the former. But the nervous system has a still more intimate connexion with the heart. Mental perturbation may actually excite organic heart attacks.

CASE III.—An obese woman with post-rheumatic aortic regurgitation dating from her youth, suffered from very occasional attacks of hyperacute pulmonary œdema during the last seven years before she died, over the age of seventy. She awoke in one attack the night after the day she received a telegram stating that her favourite grandchild was ill.

CASE IV.—Another woman suffered from frequent severe and typical Stokes-Adams attacks, which were, as a rule, excited unquestionably by mental disturbance.

I have described elsewhere cases of coronary occlusion occurring the night after some unusual emotional stress,[7] and the relationship of conflicting emotions to the onset of anginal attacks has been recognized since Heberden. The close connexion of the heart with the nervous system is reflected in the use, since antiquity to the present day, of the same word to signify either the sources of the emotions and mental tendencies on the one hand or the heart on the other. This confusion is to be seen in dozens of phrases and words, such as from the bottom of the heart, heartfelt, heartache, with a heavy heart, heart failing one, heart-breaking, heart bleeding for. The will to live may be the deciding factor in heart disease, as also in such diseases as pneumonia. I remember two North Australian aborigines at different times pining and dying within a few weeks for no other reason that I could see than that they were brought from their wild haunts to a town gaol. Dr. Jones, of Samarai, about twenty years ago told me he thought it necessary to operate upon a New Guinea native allegedly to remove a saucepan from his "stomach", because he was of the opinion the man would have died otherwise from the influence of "pourri-pourri" "caused by" the enemy who had inserted the saucepan. A long line of abdominal skin was incised and duly sewn up again. The patient made an uninterrupted recovery on awakening from the general anæsthetic to find alongside him a large blood stained saucepan. But the influence of mental factors on mortality seems to occur in the more complex white also, at any rate when serious heart or other disease is present. I have several times seen a sharp rebuke to a patient slipping into a mental torpor during pneumonia, at any rate definitely help him or her to put up a better fight, even if it did not contribute to the ultimate recovery. The heart of the Berlin Congress of 1878 was the indomitable spirit in the opposing bodily failing Beconsfield and Gorchakov. Captain Scott, the leader in the final dash to the South Pole, was the last to die.

The patient in Case III, although fat and restricted in her movements, lived for an exceptionally long time after the onset of the attacks of hyperacute pulmonary œdema. She was not only a lady of great heart, but she died a

month after her previously active, although anginal, husband had been lying in bed from an attack of coronary occlusion. Furthermore, he died a week later. The aged pair were very attached.

CASE IV is similar, except that Darby and Joan are still active, doing all the lighter house work between them. They had been looking forward to a trip to England after his retirement from the Government service on account of age. Meanwhile her Stokes-Adams attacks appeared, but in spite of these they accomplished the visit successfully, and since then he has weathered an attack of coronary occlusion eighteen months ago, which, as a matter of fact, occurred the night after she had a particularly severe Stokes-Adams attack. Incidentally, long courses of ephedrine have helped her considerably during the latter period.

Moreover, heart disease is especially dreaded, and fear, a normal emotion, may at times provoke an abnormal psychopathological complication. Again, heart disease may cause mental exhaustion, especially from want of sleep, so that it may become complicated by neurasthenia. The influence of nervous factors is to be carefully weighed in every case. It often will be apparent whether the nervous system be important or not after the patient has uttered a few sentences, apart from whether organic heart disease be present or not. But the decision in some cases whether symptoms are nervous or not can be remarkably baffling, and it is the confusion introduced by the nervous system that makes a lengthy investigation often necessary. Lastly, once the decision be made that certain symptoms are neurotic, it is most important to attempt to differentiate between the two nervous ætiological groups:

(12) Fatiguing stress, whether from constitutional disease, from mental disturbance or from the environment (resulting in neurasthenia).

(13) Psychopathological conflicts (resulting in a psychoneurosis *et cetera*, such as hysteria).

Paramount Importance of the Pumping Muscle.

The function of the heart is to act as a pump. Disability in heart disease comes only when the heart muscle pumps insufficiently. The ventricular heart muscle is the main part of the engine. The most important aim in our diagnosis therefore is to estimate the extent of crippling of the ventricular myocardium, actual or potential. Faults in the distributor, carburettor, valves or lubrication system will threaten to interfere with the action of pistons and cylinders. So likewise may potential exhaustion of the heart muscle lie, more or less respectively, either in disease of the junctional tissues, the vessels or valves, or in the prevention of adequate stroke from the seizing of pericardium by adhesions between its own layers and the mediastinum.

The Heart Muscle Paradox.

Permanent serious breakdowns of the heart muscle do not, as a rule, occur if the auxiliary parts of the engine are healthy. The heart muscle has the most marvellous power, not only of endurance, but also of recovery, provided it be adequately nourished. Death is rare from disease of the heart muscle only. Indeed, the myocardium is as a rule immune to the toxins of most infections. The only natural disease in which death from failure of the heart muscle from direct poisoning is at all common, is diphtheria. Death from acute myocardial degeneration is common in untreated beri-beri. But beri-beri is not a natural disease. It is due to man tampering with his natural food. The heart has been extraordinarily well selected during evolution, and this applies especially to the pumping muscle. The permanent recovery of this muscle in such cases as Case II and in diphtheria exemplifies this.

CASE V.—I saw in consultation a pregnant young woman with complete heart block, probably due to an attack of diphtheria in childhood, who went through her confinement without the development of any cardiac symptoms. Although the comparatively tiny auriculo-ventricular node had apparently become permanently damaged, the pumping muscle had fully recovered. (The electrocardiogram was normal, except for the complete dissociation of the auricular and ventricular rhythms.)

The arterio-capillary type of arteriosclerosis commonly associated in the kidney with hypertension and to a less degree in other organs, is, according to Evans or Fishberg, rare in the heart.[7] (This is different from that type of arteriosclerosis of which coronary sclerosis forms a part.) Even extensive fibrosis within the heart muscle itself does not cripple it *per se*.[7] The cause of the fibrosis may be still operating and be a menace to the myocardium, as is the case in coronary sclerosis. The fibrous tissue, however, is a monument commemorating a victory, although at the same time marking the grave of muscle annihilated at that time. The power of recovery of the heart muscle is seen in rheumatic fever, not only following the condition of acute myocarditis, but especially afterwards, in the manner in which it combats the strain of crippled valves by hypertrophy. It is amazing to consider how the heart muscle must have carried on in life when we contemplate the extraordinarily advanced degrees of either mitral stenosis or aortic regurgitation that are occasionally found *post mortem*. Post-rheumatic valvular disease is common, but the cases in which the lesion becomes so far advanced as to defeat the muscle without the aid of some other factor are not common. The heart muscle is often known clinically to carry on with defective valves for half a century and more, especially in those cases of aortic regurgitation which escape subacute endocarditis, as, for example, Case III. Patients with mitral stenosis are much less likely to develop subacute endocarditis, but much more likely to develop auricular fibrillation. Adhesions between the pericardial layers and between these and the mediastinum, so advanced as to hamper seriously systolic contraction of the ventricles, are likewise fortunately still more rare, and even then, as with valvular disease, will the heart be defeated only after fighting every inch of the way with increasing hypertrophy. The number of cases of arrhythmia which defeat the heart muscle when uncomplicated by disease in the ventricular myocardium or elsewhere is negligible. Hypertensive heart disease consists of enlargement[8] due to hypertrophy and dilatation during the course of

hyperpiesis. Hypertrophy here again is the reply of the muscle to stress. *Per se*, hypertrophy does not make the condition of a heart any more serious (Allbutt). Also, dilatation, as shown by Starling, is fundamentally physiological, namely, to produce an increased systolic stroke, although, like some other useful defences of the body, such as abscess formation[5] or repression, it may at times get out of hand. Dilatation has suffered a lot of unmerited abuse, and it is interesting in this connexion to recall a statement of Starling that dogs' hearts failing to contract as a result of exhaustion during experiment can be given successive new leases of activity by successive slittings of the pericardial sac permitting greater and greater dilatation each time. In essential hypertension, although with dilatation the heart has to perform more work, the associated hypertrophy will but emphasize the supreme vitality of the heart muscle. There is, however, one important proviso, namely, that the heart muscle be adequately nourished. Heart enlargement due to hypertensive disease no doubt tends to incline a prognosis to become more serious because of the new "set" due to dilatation under some past stress becoming permanent (as well as hypertrophy). The new mechanical stresses associated with permanent dilatation no doubt necessitate the expenditure of relatively as well as absolutely greater energy during systole. The myocardium thus operates on less margin than in a heart without dilated chambers. Permanent dilatation therefore tends to become a potential risk to the myocardium in the event of some future weakening of the heart muscle, even, for example, from influenza, the effects of which are trivial on the heart under normal circumstances. But if the myocardium be not poisoned by syphilis nor otherwise hampered, as by inadequate nourishment, clinical experience shows that as a rule it will insure the breakdown to be but temporary. The main reason why the finding of heart enlargement due to hypertensive disease tends to incline a prognosis to become more serious is that essential hypertension, clinically proved instrumental by the sign of enlargement, besides causing such enlargement, also accelerates the production of that kind of arteriosclerosis of which coronary sclerosis is a local manifestation. It is the presence of coronary insufficiency due to coronary sclerosis which is the main factor in increasing the prognostic gravity in hypertensive disease.[7] Healthy coronary arteries can supply the additional nourishment required for the increased bulk of hypertrophy or the greater systolic energy demanded by dilatation. A given degree of coronary sclerosis may be present in which the blood supply is sufficient to nourish the heart muscle in the absence of either increased bulk or dilatation, but insufficient in the presence of hypertrophy and dilatation.[7] Coronary sclerosis can occur without essential hypertension, and, indeed, many cases of acute myocardial infarct due to coronary sclerosis are not associated with previous essential hypertension. However, whether coronary sclerosis occur with or without hyperpiesis, it is the same disease and liable to cause coronary insufficiency with its important complication of under-nourishment of the heart muscle. In coronary insufficiency without hypertension, the myocardium has to contend, at any rate in the first place, before any dilatation has occurred, with under-nourishment only. It can be seen that, although it is the health of the heart muscle which is the most important economic factor in the heart, this in turn is in the main dependent upon the integrity of the auxiliary anatomical structures of the heart. Unfortunately, unlike the more serious degrees of valvular disease, failure of the muscle from hypertensive disease and coronary sclerosis is very common. Although hypertensive disease is the commonest heart disease causing morbidity, it is to be recognized that even here coronary sclerosis is the important underlying factor in hastening myocardial insufficiency.[7] However, although coronary sclerosis is the clinically obvious factor in precipitating failure in those cases of hypertension in which occurs an acute coronary occlusion, it is not the obvious clinical factor which precipitates a more or less insidious onset of congestive failure, the more obvious precipitating factor often being an infection. Nevertheless coronary sclerosis is present in more or less degree.[7] Coronary sclerosis is therefore of the greatest importance in considering the general subject of chronic heart disease.

Twofold Course in Coronary Sclerosis.

Very advanced grades of coronary sclerosis, even with occlusion, at times may be present without affecting the nutrition of the myocardium. On the other hand, one small fleck of atheroma may be the cause of a myocardial infarct and even sudden death. The reason for this apparent anomaly lies largely in the collateral circulation of the heart.[7] Anastomoses exist not only between the right and left coronary arteries and between the branches of the same artery, but also between the Thebesian channels and the coronary vessels. The anastomosis tends to develop with the onset and progression of coronary sclerosis. It is said to become more open with advancing years also. Whether this be so or not, there is considerable variation in its development in normal hearts of the same age.[7] Furthermore, arteriosclerosis is characterized both by the variability in the rate of its progress and the capriciousness of its distribution, the lesions being either more proximal or more extensive in some hearts than in others. Accordingly, therefore, whilst the anastomosis in one case may sufficiently keep pace with the advance of coronary sclerosis to prevent coronary insufficiency, coronary insufficiency in another case may outstrip the development of the collateral circulation. Cases approximating to the former group either never develop myocardial insufficiency, or if there should develop slight degrees of coronary insufficiency, myocardial insufficiency will tend to be gradual. On the other hand, marked degrees of coronary insufficiency, due

to bad luck in anastomoses and in "nipping" of a coronary lumen more or less proximally, tend to cause sudden overwhelming (a blighting rather) of the myocardium by occlusion. Time is the important factor for the development of the anastomosis. In acute coronary occlusion the time factor is suddenly swept away. The diagnosis of the time factor is accordingly of the utmost importance in any case of coronary sclerosis. Clinically, therefore, an attempt is to be made to divide cases of coronary sclerosis into two groups: on the one hand, those cases in which the diseased artery or arteries are more or less in the proximity of becoming insufficient to supply the corresponding focal area or areas of heart muscle; on the other hand, those cases in which there is not this proximity. Coronary insufficiency is relative only. A coronary artery may be carrying sufficient blood to nourish its area of heart muscle when the individual is walking on the flat. But it may be insufficient on a hill. In the latter case warning of the insufficiency is sometimes, but perhaps not always, given by an attack of anginal distress. The same kind of warning of insufficiency is sometimes given in an abnormal spasm reflexly resulting from conflicting emotion or contact with cold or possibly at times from digestion.[7] The pathological lesion in the heart muscle associated with an anginal attack from coronary insufficiency resulting from sclerosis is but a temporary condition of ischæmia, which in a later attack may become permanent as an infarct.[7] Proximate coronary insufficiency[7] is the name used to describe the condition present in those cases of coronary sclerosis in which the myocardium is in danger of becoming suddenly overwhelmed by occlusion. The other conditions in which anginal attacks occur are syphilitic heart disease and aortic valvular disease, especially regurgitation. Incidentally it is to be noted that, owing to the collateral circulation, the area of an infarct resulting from coronary sclerosis is less than the focal area supplied by the arterial branch. Fortunately anginal distress gives a warning in many cases, but an infarct is often not heralded, this being then the first evidence of coronary insufficiency. Fortunately for the practitioner in a hard world, the great majority of patients who seek advice before a myocardial catastrophe occurs have had premonitory symptoms.

Clinical Methods.

The foregoing are only principles in the mental background which consists of our whole knowledge, not only of heart disease, but of medicine generally. The foreground of a heart investigation likewise consists of many clinical methods, but here again there are a few outstanding methods. Far and away the most important is the taking of the history. The next is examination by the electrocardiograph. These two methods are in a class by themselves. Their findings are closely connected in the important clinical condition of coronary sclerosis. The history informs us of the presence of proximate coronary insufficiency, the electrocardiograph of the degree of myocardial involvement. The electrocardiographic findings must always be tempered by the history because of the not infrequent menace to the myocardium of proximate coronary insufficiency. Estimation of the size of the heart occupies third place. This is important because hypertensive disease is the commonest cause of cardiac invalidity. In the vast majority of cases the size of the heart may be estimated by the ordinary clinical methods of palpation, percussion, and last, but by no means least, auscultation with a bell stethoscope. Next is the determination of the presence and nature both of valvular disease and arrhythmia. In the great majority of cases either can be determined by auscultation. The electrocardiograph will occasionally be necessary for both arrhythmias and valvular disease, and so also may the oscillometer be found quite useful, especially for *pulsus alternans*. The estimation of blood pressure comes next, and so also does the examination of the retinal vessels. Examination of the pulse at both wrists, the cervical veins,[4] the lungs, the liver, the subcutaneous tissues, the urine, especially the amount, and sites of possible focal sepsis are all important. The ascending aorta may sometimes require screening, mainly for the purpose of investigating the presence of syphilis, and occasionally the size of the heart cannot be estimated except by X rays, owing to the presence of obesity or extracardiac disease within the thorax. The X rays may be useful also as well as Duroziez's test (by the electrocardiograph) to confirm the presence of fixation of the heart within the chest by mediastinal adhesions. The temperature chart may be of the greatest importance to confirm the presence of acute coronary occlusion, and sometimes the leucocyte count may be useful for this purpose also. Investigation by the Wassermann or similar test, by the blood count or by estimation of the basal metabolic rate may be found often advisable.

The history remains of greatest importance in the investigation of a possible heart case. The condition apparently may be revealed in the complaint, but full inquiry is generally advisable, especially of the circumstances surrounding both the symptom at onset, the sensation which led the patient to believe he was ill, and associated symptoms. The history, namely, the presence of anginal attacks, alone can inform us of the presence of proximate coronary insufficiency, although the electrocardiogram, the retinal arteries or the second aortic sound may make us suspicious. The second aortic sound is raised in pitch in syphilitic aortitis, in atheroma, and in raised blood pressure, but it is often raised in neurasthenia also. The characters are somewhat different respectively, but the ordinary ear cannot be relied upon to make the distinction. Furthermore, the history is necessary in a large degree to estimate ætiology. For example, a history of frequent attacks of tonsillitis or of quinsy may sometimes be a better guide to the

presence of tonsillar sepsis than the appearance of the tonsils. A history that "nerves" have been removed ten years or so from teeth may point to the necessity for an X ray examination. As the Wassermann and other blood tests have been said to yield no reaction in something like a third of cases of cardiac syphilis, we may have to rely on the history if signs be anomalous or if it be either impracticable or unwise to make the therapeutic test without clear indications. The history may be our only method available to enable a diagnosis of paroxysmal tachycardia to be made. The severity of the exciting cause of a cardiac breakdown and the results of treatment are both important in estimating a prognosis. The functional efficiency of the heart as a whole is given by the history of the response to effort, especially in relation to the daily routine. This is a more generally useful guide as to how the heart is carrying out its work than exercise tests. There are important fallacies, however, in both. An apparently poor functional response may be found in that form of neurasthenia which has been labelled "effort syndrome", and yet the heart in this condition is virtually normal. A poorer response to effort than is warranted by the state of the heart muscle may be given when the patient has allowed himself to become unfit. On the other hand, there may be a history of normal response to effort, thoughbeit very seldom, when the heart is by no means normal, and even manifest by other clinical methods to be in a potentially serious condition.

CASE VI.—An unusually fit, well grown man of forty-two, who had always made a point of keeping himself in condition by athletic exercises, 177·5 centimetres (five feet eleven inches) in height, came under the observation of his medical attendant because he was refused an insurance policy in 1930 on account of raised blood pressure. He was referred to me in 1932 and it was unlikely that such rejection was a motive for the statement then made that he suffered from no symptoms except that when he ran a hundred yards he would become "puffed". Moreover, his medical man, a most careful physician, stated: "he can perform a great deal of physical exercise without distress", and he was playing good class cricket without any disability. The heart was enlarged, but not excessively, the point being in the fifth left interspace three-quarters of an inch outside the nipple line. The blood pressure, however, was 280/162/155 millimetres of mercury (with Pachon's new oscillometer), the retinal arteries were sclerosed and the electrocardiogram showed a convex *S-T* interval leading to a diphasic *T* wave in Lead I (see Figure I). There was no history of any previous cardiac breakdown. It is a point of incidental interest in this case that his doctor reported that the patient could alter his blood pressure, especially his systolic, at will. He performed the trick for me; with intentional mental stress the pressures rose to 320/168/160 millimetres, but he brought it back to 220/160/152 within a few minutes by, he stated, "shutting my eyes and emptying it (*sic*) out, clean out, trying to go to sleep, or keep on saying one word and sleepingly". It can be seen that there was unquestionably a great difference in the systolic pressure, and I am of the opinion that the slight alterations in the mean and diastolic pressures respectively were accurate.

Although the psychoneurotic may at times exaggerate his symptoms, he may, on the other hand, withhold a poor response to effort more or less unconsciously from repression. The history of fatigue, of habits, including exercise, or of gastro-intestinal and psychological symptoms, especially insomnia, is important, particularly from the point of view of treatment.

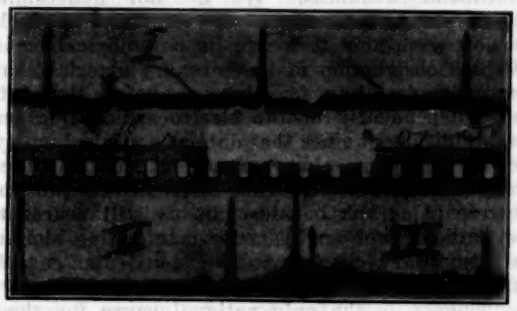

FIGURE I.

The size of the heart is important. Chronic enlargement of the heart from hypertrophy and dilatation indicates that the heart muscle has been subjected to strain in the past. In the absence of either valvular or pericardio-mediastinal adhesions, it indicates present or past essential hypertension as a rule, but not invariably. However, the size of the heart is of help in prognosis in some cases only. If the heart be very large, the prognosis is generally serious (although not invariably). But a slight or even moderate grade of enlargement is not necessarily serious. Moreover, a heart may show no enlargement and yet be in a serious state, either from the condition of the arteries or myocardium.

Neither the history nor the size of the heart can provide us with an estimate of the state of the heart muscle. At the London International Congress just two decades ago, no less a quartet than Allbutt of England, Vaquez of France, Wenkeback of Germany, and Thayer of America deplored the absence of any clinical method to measure cardiac insufficiency, whilst Cabot at the same period wrote that the diagnosis, letting alone prognosis, of what at that time was called "chronic myocarditis" was purely a matter of luck. It was recognized that patients sometimes recovered from the severest grades of congestive heart failure, even in hypertensive disease with a normal rhythm, and furthermore, might carry on then for years without any symptoms of heart failure, as, for example, in a case described elsewhere.[7] It is easy to perceive afterwards that the heart muscle could not have been permanently seriously damaged. But at the time the heart was obviously labouring, it was impossible to know, in the absence of any clinical method to estimate the myocardium, that it was but temporarily overcome by a combination of circumstances, as, for example, in that case by intercurrent influenza associated with fatigue occurring during hypertensive disease. It was also recognized that angina may be the precursor of a serious myocardial catastrophe. But, although the opinion was widely held, from Jenner to Mackenzie, that many cases of *angina pectoris* were caused by

coronary sclerosis, conceptions as to myocardial and coronary insufficiency were so confused that potential was not distinguished from actual myocardial weakness. We do not understand angina yet, but, thanks to the electrocardiograph, we now seem able to locate its pathological basis. "Sudden obstruction of the coronary arteries" had been recognized by a few observers before that date and independently of the electrocardiograph, but its establishment since that date as a clinical entity, and by no means always a serious one, has been brought about through this instrument. But the electrocardiograph has brought us still nearer to the pathology of angina. By manifesting similar but temporary changes in the ventricular muscle during anginal attacks, it has shown that coronary insufficiency is the only rational cause for those cases of *angina pectoris* which occur in patients suffering from coronary sclerosis.[7] The more or less definite conception of proximate coronary insufficiency has thus become possible since that date with consequently clearer conceptions of not only how, but also when the myocardium becomes involved. In the period before any permanent myocardial involvement we cannot expect to find permanent myocardial involvement *ipso facto*, although proximate coronary insufficiency may be present already with its serious potentialities. Evidence of potential but not yet actual involvement of the myocardium must necessarily be sought for outside it. Proximate coronary insufficiency is one thing and myocardial insufficiency another, although the latter is commonly caused by the former. It was also recognized that the response to effort might be by no means poor and yet the myocardium be apparently seriously involved already, as in many cases of beri-beri. This fact has become still more emphasized since the use of the electrocardiograph, as in Case VI. Occasionally the myocardium is even so diseased that death is imminent, and yet the complaint may be trifling.

CASE VII.—I have described elsewhere a patient with auricular fibrillation who made light of his only symptom, epigastric pain, and insisted on working and boating. But the electrocardiograph revealed that most serious picture, so-called arborization block, and he died comparatively suddenly two weeks later.[8]

It is the electrocardiograph which has provided us with the one definite method we possess for estimating the health or otherwise of the heart muscle. Without the electrocardiograph there are only three reliable signs of serious disorder of the pumping muscle, namely, "cardiac asthma", *pulsus alternans*, and gallop rhythm. But the myocardium is often seriously affected without any of these signs being present. Furthermore, although cardiac asthma is a fairly common symptom of serious disease of the heart muscle, the majority of patients dying within two years, nevertheless a considerable number live on for five or more years after it has made its appearance. Indeed, it is the electrocardiograph which often, but not invariably, enables us to give a prognosis as to the expectancy of life in cardiac asthma. The presence of auricular fibrillation or of complete heart block is often used to indicate that symptoms complained of are produced by disease of the ventricular heart muscle. These disorders are, of course, due to heart disease, but they are not due to involvement of the ventricular myocardium and, if this be healthy, not only is little or no inconvenience felt, especially in the case of heart block, but the cardiac prognosis is by no means serious (see Case V). Again, I have seen in Queensland several cases of dyspnœa due not so much to the auricular fibrillation present as to less clinically obvious coexisting silicosis. The electrocardiograph is invaluable in such cases, and, indeed, may be the means to put us on the right track. Weakness of the pulse or of the first heart sound has been cited as a sign of weakness of the myocardium. Both are so fallacious as to be often misleading, and must not be used. The only times when a weak first sound might prove useful is when it appears under circumstances in which we suspect either an acute myocardial infarct or pericardial effusion, when it happens to be known to have been loud previously. In the respective circumstances a weak first sound may help to clinch a diagnosis. Innumerable methods have been brought forward for estimating the heart muscle in chronic cardiac disease, especially before the use of the electrocardiograph, but all have been proved useless for this purpose except that instrument. The electrocardiograph is not infallible. For reasons already given, it does not help us in the diagnosis of uncomplicated proximate coronary insufficiency, the condition in which lies the grave possibility of the myocardium becoming suddenly ruined by acute coronary occlusion. But it does provide us with a method of discovering when the ventricular muscle has become seriously involved, whether by syphilis or by more or less insidious coronary insufficiency, as well as with acute myocardial infarct. There are only rare exceptions to this, and in such exceptions, so far as I am aware from my own experience, some other evidence, such as cardiac asthma, will usually be found.

Although the electrocardiograph is for practical purposes diagnostic of ventricular muscular involvement, it is not so useful in prognosis, although it has a definite place which cannot be filled by any other method at the present day. The reason why the electrocardiograph is not of the same value in prognosis as in the diagnosis of involvement of the ventricular myocardium is that this muscle is fairly commonly overwhelmed suddenly by acute coronary occlusion. Sudden death may occur, or, if the patient survive the attack, coronary sclerosis is also more or less present in the remaining coronary branches in the great majority of cases. Proximate coronary insufficiency therefore may exist still. Not only, therefore, may the extraordinary power of recuperation of the remaining heart muscle tend to be hindered by insufficiency both of the coronary bed generally and of the collateral circulation (which, however, is developing meanwhile, especially after the attack), but the potentiality for

another catastrophe still may exist. Clinically we do find a few cases in which the patient remains free of symptoms for years after recovery from an attack of acute myocardial infarct, but we cannot afford to ignore the possibility of another attack in such cases. They must be labelled accordingly as being still cases of proximate coronary insufficiency. If we possessed an infallible method, whether by the history or otherwise, of foretelling the occurrence of acute coronary occlusion, the electrocardiograph would give us a fairly accurate prognosis of the myocardium in the remaining hearts. Unfortunately we have not. Proximate coronary insufficiency may be present and yet no symptoms, as well as no adequate signs, be present before the onset of an attack of "acute occlusion". However, especially in patients who seek medical advice, the attack of permanent focal injury to the myocardium is generally heralded by prodromata consisting of temporary ischæmia manifested as attacks of angina. However, not only may the history of these be difficult to interpret, but they may not be easy to obtain.

CASE VIII.—I was called in casually to see a fit but nervous little man of fifty-five because a nurse friend had advised it, and there had been no suggestion that he might be suffering from heart disease. He complained of being off colour for six days, of his stomach swelling, and of sore, running eyes. He had stayed in bed that morning only. He had had a three months' illness eighteen months before, when a capable clinician wanted to remove "gall-stones". He seemed to me at first to be suffering mainly from fright, namely, that he would have to have a gall-bladder operation. It was only after I had found raised blood pressures (200/131/125 millimetres of mercury with Pachon's new oscillometer), nipped retinal veins, and a *T* wave in each lead of the electrocardiogram typical of a focal myocardial lesion, that I managed to secure a history that he had felt slight discomfort in the epigastrium at times when he had over-exerted himself since the severe illness. On reviewing the history of that illness he stated that it had commenced with intense giddiness in the street, that he vomited for two days, that there had been a temperature never above 37·8° C. (100° F.) during the few days following the onset, and that the doctor kept getting him out of bed but had to keep putting him back again during the first three weeks. Moreover, the pain is now said to have been more a distressing feeling of fullness at the upper part of the abdomen. Although the symptoms of acute coronary occlusion were thus also atypical, there is no valid history of gall-stones. He has had more definite attacks of angina since, tightness having been felt about the throat when he has over-exerted himself, and also at other times.

Although one should not rely on the electrocardiograph in suspected cases of proximate coronary sclerosis, this case shows how valuable that instrument may be at times.

Leading questions must, of course, be avoided if possible, but as the history is our only clinical method of diagnosing the important condition of proximate coronary insufficiency before any permanent effects have become manifest in the heart muscle, it is necessary to ask leading questions concerning pain and other distress. When dealing with suggestible patients, leading questions should be, of course, couched in the negative. Inquiry concerning other symptoms besides pain may bring forth a history of anginal attacks. A question concerning attacks of difficult breathing not only may elicit information concerning paroxysmal nocturnal cardiac orthopnœa ("cardiac asthma"), but attacks of suffocation may then be brought to light. I have found inquiry concerning the word "indigestion" occasionally fruitful when more direct questions have failed to elicit the presence of angina. The great majority of pains about or near the chest are easy to differentiate from the point of view of angina or not angina, even when the attacks are not distressing and do not conform to the classical description of *angina pectoris*. The main desideratum is to realize that a mild attack, such as slight discomfort about the chest or epigastrium or awakening with suffocation, may be just as much an anginal attack as the attacks described by Heberden, especially in a patient beyond middle age. It is easy to recognize angina in a more or less transient suffocation or tightness felt at the sternum, single and isolated from other symptoms either during or between the attacks, except that it is intermixed with greater or less degree of a sense of ill omen, consistently startling and arresting the patient each time he walks up some particular rise. But some cases are difficult to distinguish, and an occasional case is impossible except by more or less prolonged observation. An isolated pain may at times give difficulty, as, for example, in fibrositis or gall-stones. However, when we are in a quandary there is generally present a multiplicity of symptoms to which proximate coronary insufficiency may be contributing. Coexisting disorder may be either above or below the diaphragm, but the most confusing histories are obtained from cases in which angina is embroidered with pain in the region of the chest, due to either neurasthenia or a psychoneurosis. It is the possibility of a neurosis being present along with angina which precludes diagnosis by exclusion.

Another difficulty in differentiating between angina and neurosis is that both are investigated by the history and, as is well known, not only may neurotic pain mock angina, but anginal attacks may be atypical. There is no symptom or group of symptoms to which angina will always conform. The anginal attacks may have seemed so slight and trivial to the patient that he has ignored them. On the other hand, attacks of precordial pain due to nervous causes may so resemble atypical cases of angina or even sometimes mimic more or less typical angina, that diagnosis becomes very difficult. The more experience I have had of these cases, the more I have become convinced that there is no royal road to differential diagnosis. There is one way, however, a plodding, labouring way, that almost invariably leads us to a correct conclusion and must be trudged, namely, a lengthy and patient history taking. As both angina and the neuroses are essentially recognized by the history, a main approach can come this way only. Other methods of examination cannot be substituted, although they should be mobilized to help. The investigation is tedious. Relationship to anger or conflicting

emotions is characteristic of either angina or psychoneurotic attacks. Tightness or suffocation is characteristic of the anginal attack, but all anginal distress is not felt as a heaviness, pushing, constriction or suffocation, and occasionally, on the other hand, a neurotic patient complains of a gripping pain in the chest or of suffocation in the throat. Bizarre sensations are occasionlly ascribed to anginal attacks. On rare occasions pain which is probably anginal is said to have been sharp. This may be not only because of a patient's poor powers of description, but also because anginal distress is sometimes indescribable. Angina at times occurs in the region of the left breast, as well as the epigastrium and the hand. On the other hand, discomfort in neurasthenia may occasionally be located about the sternum, and pain, numbness or deadness is sometimes described in a neurosis as having radiated to the arm. Neurotic patients may state they awake with suffocation or pain, and it cannot always be elicited on further inquiry that they had not really fallen asleep. Moreover, anginal attacks may at times be excited by dreams. A psychoneurotic, if he has possessed previous knowledge, may tell a tale characteristic of anginal attacks. But, so far as I am aware, I have had no experience of a neurotic pain in which anginal characteristics were combined with absence of other pains or symptoms either during or between attacks. Nevertheless, the same difficulty exists in hysterical counterfeits of angina as in those neurotic patients in whom atypical angina may be present also, namely, how is the case to be clinically decided? An analysis of symptoms may sometimes suffice to differentiate the symptoms of proximate coronary insufficiency from a neurosis, but as a rule it is essential to investigate carefully the onset of invalidity. Inconsistency and vagueness are characteristics of neurotic symptoms. However, genuine cardiac patients may at times be vague concerning angina or other heart symptoms either from stupidity or repression; whilst, on the other hand, one common type of psychoneurotic attacks is characterized by consistency, namely, attacks in which the patient, though unaware, lives again some past experience. Unconsciousness is more likely to be prominent in this type of hysteria than pain, but occasionally pain in the chest may be more or less prominent, as in Cases IX, XI and XII.

Just as it is necessary to exclude positive evidence of anginal attacks in a neurosis, it is also necessary in cases which might be atypical angina to exclude positive evidence of the neuroses. This is to be searched for not only in symptoms but also in causes. Evidence of fatiguing stresses before the onset may point to neurasthenia. A psychopathological cause for the pain may be inferred if not only environmental circumstances have been such as to warrant the onset of psychogenetic disease, but factors related to this were associated with the onset of the pain also. It is of the greatest importance to hear the circumstances of the first attack of distress and this should always be sought for. Indeed it is this, far more than the analysis of symptoms or even the circumstances related to the onset of different attacks, which is our most reliable guide to differentiating between anginal and neurotic pains. However, a characteristic of the psychoneurosis is repression. Accordingly, not only has the psychoneurotic patient often forgotten the early attacks, but he may be under the impression that some later attack is the first. One must submit to patient listening, more or less apparently casual, although guiding questions are occasionally interpolated. The patient may then suddenly become aware of long forgotten attacks of precisely the same pain many years before. Finally, it may be seen that the pain (and similarly for other symptoms) was originally so linked up with some environmental circumstance that the diagnosis of psychoneurosis is for practical purposes certain, provided there be no positive evidence of heart disease. But furthermore, even if heart disease be present as well, this cannot be held to be causing any given symptom unless not only is the latter of a nature and degree compatible with the heart lesion, but it also must not be a symptom which can be shown to have been brought on as part of the psychoneurosis.

CASE IX.—A well developed, big, healthy, self-assured man of forty-five, following the same profession now as when he was a non-combatant officer during the war, complained of attacks of dull pain, generally prolonged for two hours or more, about the left mammary region and a tiredness in the left arm, as well as breathlessness on exertion, all of which he, in the first instance, stated dated from the time he was "gassed by phosgene". It transpired later that he was not evacuated for phosgene, but because of blindness due to mustard gas. He became convalescent and was about to be sent back to France when he fainted whilst walking in the hospital grounds. He was put to bed again, labelled as a "heart case", and then the theory of phosgene gas was advanced to explain his alleged cardiac condition. The attacks of pain developed during this period (he was in a ward amongst many other patients with medical conditions) and at the same time he also developed "breathless attacks", which have been repeated in the early hours of the morning, exactly the same, but at long intervals since. From a London hospital he was transferred to Harrogate and was on his way by train from there to France when he fainted again. He stated that he had become unconscious three times since he left the army, volunteering: "I know it was the result of extra physical exertion" each time. No attempt was made to send him to France again, but after being told he must always avoid any strain, he was invalided out as a "heart case", since when, to use his own word, he has had "trouble". He has sniffed "amylnitrite" regularly in the intervening fourteen years, and it is interesting to note that the blood pressure (Pachon's new oscillometer) was only 150/95/90 millimetres of mercury, his heart was not enlarged, the pulse rate was 76, and his retinal arteries seemed perfectly healthy. Although there is the slightest degree of want of alignment of the *S-T* interval with the base line, I would not call the electrocardiogram abnormal to a degree which might be regarded as considerable, so that, if the electrocardiogram, for the sake of argument, should be held to be abnormal, it could not be held to be related to the above symptoms (see Figure II). A bridge inserted before the war was still present when he was referred to me for an opinion in 1932, and he complained also of fibrositic pains and tiredness since a recent attack of influenza. Focal sepsis might perhaps be responsible for the slight alteration of the *S-T* interval. My opinion to his medical attendant was that, whilst the teeth should be examined

by X rays and dealt with, the major syndrome was an independent hysteria.

Fainting from hysteria is common, and accordingly attacks of unconsciousness from hysteria are not uncommon, intercurrent with, but independent of, valvular or other cardiac disease. Unconsciousness does occur in heart disease, but under definite conditions and only very occasionally, namely, in Stokes-Adams attacks in complete heart block, at the onset of an attack in a few cases of paroxysmal tachycardia, rarely from sino-auricular block, in a rare case of acute coronary occlusion, and very rarely with very severe pain in an anginal attack.

Occasionally, in a psychoneurosis, the links with the past may be perceived in some recent symptom.

CASE X.—An undersized and war-worn youth, manly and without any tendency to decadency, fainted at a dressing station after helping to convey wounded there and woke up blind (from hysteria) three days later in a London hospital. He suffered from fainting attacks until he was cured by psychotherapy three years later. However, cardiac symptoms became so developed during the repeated shifts from hospital to hospital meanwhile that he came to be labelled "effort syndrome", and when I first saw him three years after the onset of his invalidity, his complaint was pain in the chest without any mention of fainting attacks. These became revealed later, and it was the circumstances under which recent fainting attacks occurred which were used, not for the purpose of diagnosis, but for treatment, as described elsewhere.[6]

CASE XI was a very similar case to Case X. The patient was likewise of a responsible type, but overstressed by service in the trenches. This patient, thirty-five when he was referred for an opinion in 1932, also complained of attacks of dull precordial pain; and it transpired later that these were related to fainting attacks which, however, commenced at least as far back as the occasions of two vaccinations before going overseas. It also came out later that he had two abscesses opened, one on the face and the other on the hand, just behind the lines. He fainted on one occasion, but was able to avoid this the other time by pushing his head between his knees. He was not evacuated. He also complained to the medical officer, when the latter was squeezing the hand, that it was "rough"; he could not have the hand washed. The sequence of events prior to the latest attack of pain, in 1932, also collated afterwards, was that he had been feeling well until Friday, when, coming home from work, he was nearly knocked down by a car and became very shaky. On Saturday night he came home with a headache and slept badly. On Sunday afternoon, whilst lying in bed after lunch, he fainted after reading only a few pages of Dumas's "Twenty Years After". After coming to, he suffered from precordial discomfort, shortness of breath, and sweating, and he felt he was going off again, but received considerable relief by bracing his feet against the wall, placing his head between his knees and by swirling the same hand which had been lanced on active service in a dish of water. On the fourth page of that book will be found the passage: "Comminges made his appearance, with his clothes all torn, his face streaming with blood. The queen, on seeing him, uttered a cry of surprise . . . ."

Neurasthenia may complicate heart disease. Occasionally angina or coronary occlusion on the one hand may be intercurrent with pain in the chest, on the other hand from neurasthenia or psychoneurosis or both. Such cases may be so confusing that it is sometimes impossible to be sure where to draw the dividing line between the symptoms of either group. Case XII is an example, but before considering this case the sophistry sometimes existing in the use of the term "effort syndrome" should be clearly recognized. It has been exemplified in Cases IX, X and XI how, when a given mental experience was originally associated with some more or less great mental conflict, especially at a time when the mental tissue had become weakened by long-continued fatiguing stress, it may later on become repeated as a psychoneurotic symptom. The symptoms of neurasthenia may likewise become more or less perpetuated as psychoneurotic symptoms long after the neurasthenia, in the strict use of the word, had become cured by the removal of the fatiguing stress which produced it. This is the reason why "effort syndrome", really a form of neurasthenia only, seems at first sight to have become perpetuated long after the exciting cause of the nervous exhaustion has been removed. As a matter of fact, it exists no longer, but its symptoms more or less have become borrowed by a different disease, namely, a psychoneurosis. This is not only more or less cloaked by the symptoms of neurasthenia, but also had become unleashed as a result of and during such state of nervous weakness. The clinical distinction between the precipitating neurasthenia and the succeeding psychoneurosis, partly parading in its dress, is therefore by no means obvious. Indeed, one prominent author even this year describes the two fundamental states as one, namely, under the caption of "effort syndrome". Further confusion exists in that neurasthenia may complicate psychogenetic as well as organic disease, especially when insomnia is causing exhaustion. Whilst it is important to recognize the part played by continued stress and exhaustion in the shaping of symptoms, nevertheless adequate treatment cannot be initiated until the difference between neurasthenia and psychogenetic disease becomes perceived. The story of such patients is often diffuse and confusing, even when the neurosis be not intercurrent with symptoms of some organic lesion. When neurotic and anginal pains both are present the clinician must be very wide awake. The following is a case of acute coronary occlusion intercurrent with psychoneurotic precordial pain. The patient was invalided with "effort syndrome" in 1917 and from the records he seems to have been a genuine case. The conception of a neurosis became rightly perpetuated in the records, unlike Case X, but Case XII happened to be one of those exceptional cases in which an insidious organic factor was operating also. Though it is now doubtful of what the patient complained at the time, one medical officer, years after the onset of the invalidity and about two years before the attack of coronary occlusion, noted with commendable acumen that the symptoms then "resembled mild angina".

CASE XII.—A medium-sized, very active and hard-working tradesman of forty-two suffered from an undoubted attack of acute coronary occlusion in 1932, confirmed by the electrocardiograph. (The pain, however, was in the epigastrium, although a feeling of suffocation was present at the root of the neck.) I saw him first on the first day of this. He made the following statements concerning his former history. Since a time when he was gassed in 1916 he had suffered from attacks of a sharp,

heavy pain in the left mammary region just as he was dozing off to sleep. On the following day he would feel shaky. There became added to these attacks a gripping pain in the epigastrium which lasted half a minute. A feeling of fullness to the right of this was experienced also. Short stabs had been felt in the mammary region when he lost his temper and on walking. He was vague concerning when the above increment of gripping pain commenced; moreover, at a period some time after the attack of coronary occlusion he forgot about it, although he recalled it when reminded, but he was still vague concerning its onset and was inclined to date this from a period early in his neurosis. Convalescence from the infarct was interrupted by periods of irritability, depression and sleeplessness during which the apparently psychoneurotic mammary pain would return, which he still described as a "heart attack". Epigastric discomfort, generally continuous for a day or days, has occurred since, also at times so vague and inconsistent in descriptions that at first sight it seems psychoneurotic likewise and perhaps dyspeptic, but at other times so related to doing too much and relieved by rest and perhaps also by the digitalis given, that some epigastric discomfort at any rate seems probably of cardiac origin; perhaps rather of the cardiac fatigue variety than from congestion. The period when he was mentally most tranquil and least vague was that of the first days after the onset of acute coronary occlusion, a phenomenon not unusual in neurotic patients who are subject to heart attacks.

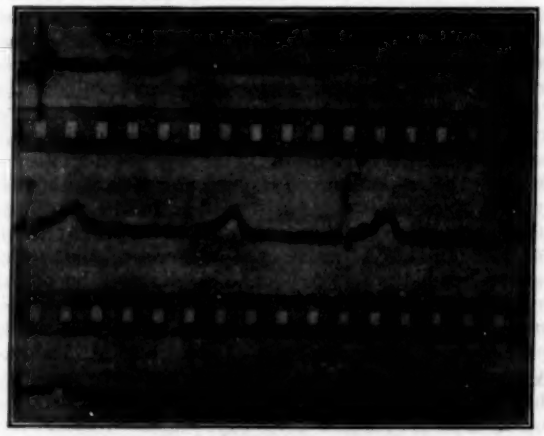

FIGURE II.

The history will at times fail to distinguish between, on the one hand, angina and, on the other hand, a neurosis or other cause, such as gastro-intestinal. As pointed out already, the electrocardiograph cannot be relied upon at a time before coronary insufficiency has produced any permanent change in the myocardium, but electrocardiographic findings manifesting involvement of the myocardium, if present, may decide the diagnosis of angina when, from the history, one has been unable to differentiate between *angina pectoris* and a neurosis. An electrocardiogram is generally useful in all cases of coronary sclerosis, even when symptoms of proximate coronary insufficiency have been present, in order to estimate how far the myocardium has become involved. Although positive electrocardiographic evidence may under certain circumstances be accepted in favour of a diagnosis of angina, negative electrocardiographic findings as a rule cannot be, for reasons seen to be obvious when proximate coronary insufficiency was considered. Nevertheless, in an exceptional case, it may be permissible at times to use a normal electrocardiogram as confirmatory evidence against the presence of angina, namely, in a case in which, if the attacks were due to proximate coronary insufficiency, the development of a focal lesion in the myocardium would have been much more probable than not.

CASE XIII.—An irascible, alert and active, but slightly podgy little business man, well preserved and fairly fit for his sixty-three years, complained of an attack of dull aching pain in the region of the left breast for two hours two weeks before. The pain was said to have been associated with nausea, pallor, cold perspiration and sufficient prostration to make him lie down in his office. He recounted a similar attack three years before, but the pain was then a "kind of a stab which came on repeatedly as if the heart were doing too much, as if valvular; when I was in insurance I used to talk about these things". He stated that he had had "eight or ten" similar attacks in sixteen years, but when he was asked the circumstances of the first attack, he then stated he did not remember, as "it may go back twenty years". The early attacks lasted only half an hour, and every attack except the last one had been relieved by a bowel evacuation, the cold sweat in those attacks occurring then. Nevertheless, he was definite that the last attack was of the same nature as the earlier ones. He was of constipated habit and required a laxative frequently, but he did not take this daily, and the motions were occasionally hard. He smoked twelve to fifteen cigars daily, as well as a pipe. Apical infection, one large granuloma being beneath a tooth dead over fifteen years, and pyorrhœa were considerable. The blood pressure was not strictly normal, being 170/105/100 millimetres of mercury (Pachon's new oscillometer), but the heart was not enlarged and the retinal arteries seemed normal. He was breathless during the first set of tennis, but not in the later three to five sets which he played every Saturday afternoon.

Such early breathlessness occurred probably, at any rate in large part, because he had had very little walking and no other exercise during the rest of the week for the preceding five years, and tennis is a strenuous, probably too strenuous, exercise for his years. The condition does not seem cardiac, but a definite decision had to be made whether he could continue to enjoy his tennis. Although the axes of *Q R S* tend to be deviated to the left, the electrocardiogram shows no evidence of weakness in the heart muscle (see Figure III). If the attacks were due to either angina or infarct, it would be very unlikely for the electrocardiogram to remain normal after so many years of apparently the same disorder.

The patient was told to regulate his bowels properly with a daily cold water extract of senna pods and to walk more during the week, as well as to have offending teeth extracted and to be less self-indulgent with his food and tobacco, but he was told he might continue his tennis.

Single electrocardiographic pictures may be unreliable for the purpose of affirming that the electrocardiogram is normal. I have seen this occur in syphilitic myocarditis; but it typically occurs occasionally when the myocardium is undergoing a change as a result of coronary occlusion. A single picture then at times may appear normal, yet a succession of pictures will show as a rule, although perhaps not invariably, the change which the ven-

tricular myocardium is undergoing. Such normal pictures in coronary occlusion may occur early in the attack, but sometimes they become sandwiched between two periods in which the inverted *T* wave has the characteristic coronary conformation. I have described two cases in which this anomaly was manifest.[7] It can be seen that if an electrocardiogram had been taken of either patient only during the period of being apparently normal and the precaution was not taken of taking another picture, the electrocardiogram would have been falsely regarded as normal.

FIGURE III.

It has been said that second attacks of acute coronary occlusion do not often occur. This is contrary to my experience. I am therefore of the opinion that second attacks are common enough to prevent us using the electrocardiograph to give a prognosis of the state of the heart muscle once there has become manifest a state of proximate coronary insufficiency. In coronary sclerosis nothing is so certain as its uncertainty. But just as probabilities are practicable in the pastime to which this epigram originally alluded, so is it practicable to give a prognosis in terms of probabilities in coronary sclerosis. Although a patient with *angina pectoris* may present a more or less normal electrocardiogram and yet a fatality may be not far ahead, nevertheless the probabilities in favour of anginal patients with a normal electrocardiogram are greater than those with an abnormal electrocardiogram.[7] If, in addition to a normal electrocardiogram, there be a deficiency disease present also, the probabilities are still further in the patient's favour. The following case is a fair example.

CASE XIV.—A medium height, spare, well developed man was forty-five when I first examined him in 1929. He had sold his blacksmith's business two years before, on medical advice, because of *angina pectoris*. This was present, but so also was pernicious anæmia. Under liver treatment he lost the attacks. Although I, of course, advised him against heavy work again, he has been back at blacksmithing for eighteen months, wielding the heavy sledge hammer. The electrocardiogram has been normal and has remained normal if one can judge from the two electrocardiograms taken since 1929 (see Figure IV). I do not see that we shall ever be able to agree to such efforts, on the theory that proximate coronary insufficiency had been present due to coronary sclerosis and was therefore now but latent.

Although the electrocardiogram has little place in prognosis in proximate coronary insufficiency, it is an important prognostic guide in those cases in which proximate coronary insufficiency can with a fair degree of probability be excluded, namely, in cases without angina-like prodromata. This statement may be exemplified by examining a series of cases of complete heart block or auricular fibrillation. The seriousness of the cases will be found as a rule to vary with the electrocardiographic manifestations of the state of the ventricular muscle. I exemplified this in connexion with auricular fibrillation four years ago.[8] Case VII of this paper was the worst case in that series. The two patients with the mildest conditions are alive and well still, although one had two attacks of congestive failure seven years ago, namely, before she was placed upon proper maintenance doses of digitalis. The electrocardiograph, even when used intelligently, is by no means infallible for estimating the condition of the heart muscle, yet, when contrasted with the findings of such useful tests as the blood Wassermann, skiagrams of the stomach and Graham's test, it not only by no means loses anything on the score of faithfulness, but it also supplies a fairly accurate degree of measurement, with certain given limitations, as discussed in this paper and elsewhere.[7]

The clinical data obtained, the diagnosis-complex may be compiled. Perspective for prognosis and treatment can be given only by this. A complex view of cardiac diagnosis will not prevent two generations of doctors so frightening patients like the patient in Case III that they come to distrust us and go to quacks, as that patient had sometimes done. It will not warn the practitioner when enough digitalis is given nor remove his prejudices in giving routine doses of morphine (another specific in heart disease), nor so make him visualize both the necrosing infarct in acute occlusion and the slowly developing anastomosis afterwards that he will first of all keep the patient motionless for three weeks, on his back if at all possible, with another three weeks at least in bed, and then advise

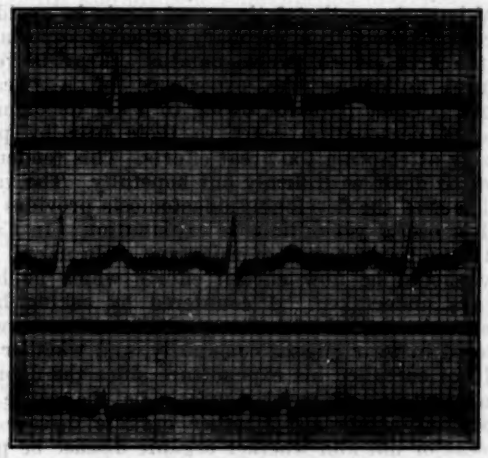

FIGURE IV.

a slow convalescence of six months or more. But a diagnosis-complex will prevent such mistakes as were made in Cases I, IX, X and XIV and the not uncommon oversight of forgetting that thyreotoxicosis may be the major syndrome, not only in association with auricular fibrillation, but also with a normal rhythm. An eye on the neurotic factor will prevent us from forgetting to treat insomnia, a potent cause for helping to bring on or prolong not only neurasthenia but also a cardiac breakdown. Incidentally I have found "Amytal" of great value for insomnia after acute coronary occlusion.

On one occasion, when an attack of rampaging mania with illusions of persecution occurred at night in an old man of over seventy, but a fortnight after an attack of acute coronary occlusion, anginal attacks having become frequent meanwhile, I secured sleep with what seemed to me at the time the alarming dose of 0·45 gramme (seven and a half grains) of "Sodium amytal". He awoke still deluded, but quiet and amenable, and has since made a remarkable recovery, being now, eighteen months later, free of anginal attacks as well as very sane.[1]

Either neurasthenia, psychoneurosis or even a psychosis may complicate organic heart disease. Neurotic symptoms may appear during the fretting restrictions necessary after acute coronary occlusion or from the alarming nature of the symptoms, and morphine may be at fault at times. I had the impression in the case just mentioned that the morphine given frequently for the repeated post-occlusion anginal attacks present in that case was in part responsible for the psychotic symptoms, in conjunction with the patient's age. Neurotic symptoms must be dealt with energetically. Nevertheless ingenuity and tact are necessary, as it is of literally vital importance that fatigue and strain be avoided in convalescence from coronary occlusion, especially in the first six to twelve months. Fear is very natural following the experience of a "heart stroke". Some patients, owing to a not uncommon type of personality, react to their fear by perversely attempting too great efforts. This tendency must be dealt with firmly but hopefully. The very real fact of the developing anastomosis provides us with a useful yet optimistic explanation. In Case VIII the patient was of this type. He asked me why should he now curtail efforts which he had always undertaken. I countered by asking him whether he argued with the eye doctor when he was ordered to wear glasses constantly, and whether he had not curtailed his expenditure since the depression started. If psychoneurosis be the cause of important symptoms, whether organic disease be present or not and whether organic disease be producing symptoms or not, it must be adequately treated. It is not "nothing".

Summary.

Adequate prognosis and treatment in heart disease can be arrived at only by means of a diagnosis-complex, but to make this as accurate as possible, understanding of our ignorance and prejudices is as necessary as up-to-date knowledge. Psychogenetic causes for nervous symptoms can be no more ignored than can be the organic causes of heart disease. The pumping heart muscle is of paramount importance, yet, possessing amazing vitality, it is overcome as a rule only because of faults in its auxiliaries. The electrocardiograph is our only clinical weapon for estimating the state of the myocardium, but it has considerable limitations which, however, in the main parallel the uncertainty of tenure held by areas of heart muscle supplied by those sclerosed coronary arteries which happen to be more or less in the proximity of becoming insufficient for the adequate supply of nourishment. Fortunately, proximate coronary insufficiency is generally, although not invariably, revealed in the history, namely, by angina. Atypical anginal attacks comprise angina just as much as those conforming to the classical descriptions. Anginal attacks in coronary sclerosis are evidence of focal myocardial ischæmia, yet but the temporary flashes giving warning of that stroke in which the ischæmia will remain permanent as an infarct.

Some clinical methods have a much more important place than others, depending partly on the value of each for some particular clinical purpose, and partly on the importance of the latter. But no clinical method is infallible, and none should be relied upon alone, especially in estimating prognosis. We should avail ourselves of all methods to our hand and then consider all the findings in conjunction. Nevertheless, clinical methods have a definite order of preference, and I place them as follows: History taking (mainly of the response to effort, particularly in connexion with proximate coronary insufficiency, and of the ætiology, especially in connexion with syphilis and rheumatic infection), electrocardiography (mainly to estimate the approximate degree of myocardial involvement, particularly in coronary sclerosis and syphilis), auscultation along with palpation and percussion (mainly to indicate the size of the heart, but also for arrhythmia and valvular disease), and the use of the oscillometer. The ophthalmoscope and examination of the sites of focal sepsis and of congestion are important also. X ray examination may be at times necessary either to diagnose syphilis by showing syphilitic aortitis, or, in the presence of extreme obesity or abnormal chest conditions, a large heart. However, there are still remaining clinical methods of more general usefulness in connexion with heart disease than X ray examination of the heart, for example, an X ray examination of the teeth, at any rate in this country in this generation, or a Wassermann test.

[1] Since this article was written this patient has had a second attack of acute coronary occlusion.

References.

(1) H. E. B. Pardee: "Clinical Aspects of the Electrocardiogram", 1928. (A new edition is in the press.)
(2) P. D. White: "Heart Disease", 1931, page 570.
(3) T. Lewis: "Diseases of the Heart", 1933, page 34.
(4) *Ibidem*, page 10.
(5) N. W. Markwell: "The Heart and the Neuroses", THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, 1926, pages 830 and 831.
(6) *Ibidem*, pages 831 and 832.
(7) N. W. Markwell: "Changing Points of View in Heart Disease", a paper read before the Medical Section of the Queensland Branch of the British Medical Association on April 24, 1933.
(8) N. W. Markwell: "The Place of the Electrocardiograph in Practice", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, 1929, pages 549 to 551.

TRICHOMONAS VAGINALIS VULVO-VAGINITIS.

By HUBERT JACOBS, M.D. (Melbourne), F.R.C.S. (Edinburgh), D.G.O. (Dublin), F.R.A.C.S.,
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MUCH literature has appeared during the last two years or so concerning the protozoon *Trichomonas vaginalis* and its associated condition, an almost specific type of vulvo-vaginitis. Although this communication adds but little to what is already known, yet no apology is offered. For the subject is of the greatest importance to every gynæcologist, and perhaps more so to the general practitioner, since the condition is of frequent occurrence in both specialist and general practice. Although the vaginal discharge is quite apparent, the associated organism very frequently passes unrecognized, and the discharge persists in spite of energetic treatment, causing the patient much misery and discomfort. The reason for this is adequately expressed by J. P. Greenhill:[1]

> There are still many gynæcologists who have not seen a case of *Trichomonas vaginalis* vaginitis, the chief reason being that they have not properly looked for the associated organism.

A case recently coming under the writer's notice aptly illustrates this.

> D.F., aged eighteen, single, but not *virgo intacta*, suddenly developed a vaginal discharge, yellow in colour, with some slight dysuria. Her family doctor instituted local treatment in the form of topical applications and douches, and had a smear examined for gonococci, but none were found. Unfortunately some colicky pain in the right iliac fossa had occurred, and in spite of the girl's protest and indignation, gonorrhœa with a salpingitis was suggested to her mother. The girl was then sent to a gynæcologist, who apparently did not alleviate the mental distress, although smears sent by him to a pathologist were reported to contain no gonococci. However, treatment was commenced, and for twelve months varied methods were adopted without any improvement. In addition, no less than twelve sets of smears were reported on in respect to gonococci, and none were found. Following this the patient was brought to me, still complaining of offensive, irritating discharge, still having colicky pain, and being markedly neurasthenic. On examining a drop of the fresh discharge under the microscope, I found numerous specimens of trichomonas among the pus cells. Diagnosis was thus established and appropriate treatment commenced, the first application being immediately followed by improvement and cure being obtained after nine treatments. The neurasthenia rapidly cleared up. Some little time later the patient was seen during an attack of pain similar to those previously mentioned, but more severe. I diagnosed the condition as appendicular colic, and on removing the appendix found it to contain two fæcoliths and a fruit seed about two millimetres in length.

The importance of such a clinical condition is thus fully amplified by the above citation, and in a brief survey I desire to offer some suggestions concerning ætiology and treatment which may be of value.

The presence of the protozoon in the human body is not necessarily pathological, as it may be found in the mouth, the rectum, the lungs, and also in the vagina, without any associated symptomatology. In the vagina it is not normally present and does not appear in the accepted descriptions of the normal vaginal flora. When it is found associated with a vaginitis, other microorganisms are always present, particularly streptococci.

The flagellate is not peculiar in its habitat to the human body, for it is present in the intestines of rats (Ratcliffe[2]); probably there are several strains or varieties, though as yet no morphological differences have been reported. However, Stein and Cope[3] report that different media are necessary for artificially growing *Trichomonas vaginalis* and *Trichomonas hominis*.

Before further progress can be hoped for in the investigation of the clinical condition, the following points require elucidation: (i) Is the trichomonas a normal habitant of the human? (ii) Does it directly cause a pathological condition? (iii) If not, under what circumstances does it thus become associated with such a condition? (iv) What is the means of transit to the vagina? (v) Is there a specific treatment, either prophylactic or curative?

In the particular form of vaginitis to be described the protozoon is always present, but whether it is associated or causative is uncertain. It is certain, however, that the vaginitis does not subside until the trichomonas has disappeared. On account of the presence of streptococci and other organisms, it is suggested that some factor, at present undetermined, produces an environment suitable for the rapid growth of the trichomonas. The rectum has been thought to be the source of the trichomonas transference to the vagina being made during the toilet. However, the trichomonas has not been found in the stools of those suffering from the specific vaginitis and also the implantation of *Trichomonas intestinalis* into the vagina has failed to produce a vaginitis. In spite of this evidence, I believe that the rectal origin is feasible, and suggest the *modus operandi* to be thus: many patients use routine vaginal douches nowadays, and for such employ enema syringes and other rubber apparatus having vulcanite nozzles. I believe that in many instances the "syringe" has been used for a dual purpose, and as it is rarely sterilized by patients, the route of transfer is obvious. A recent case of mine supports this view.

> E. McC., married, divorced and again engaged, had previously been infected with gonorrhœa and cured. Before her engagement I carried out further tests for the gonococcus; in no instance was the organism found. The patient had been in the habit of occasionally using a douche can and glass nozzle. After these tests she went away with her *fiancé* and later returned to me complaining of a profuse yellow vaginal discharge, in which I found trichomonas. On interrogation she stated that she had left the usual douche apparatus at home and had taken with her an enema syringe which she frequently used for douching.

The possible origin of the infection, as suggested above, seems to me of importance and is borne out by the additional evidence obtained in my outpatients' clinic, where most of the patients use, for vaginal douches, some such means until they are advised of the correct method.

The trichomonas has now been successfully cultured and subcultured, the best medium being serum-saline-citrate. Inoculation from such cultures

fails to produce any pathological condition, and clinically the vaginitis is not contagious. The organism is apparently never present at the same time as the gonococcus. I am able to confirm this, for I have seen cases following the cure of gonococcal urethritis and cervicitis. Indeed, the presence of a trichomonas vaginitis following such conditions appears to confirm the success of such treatment. A case of mine illustrates this:

M.F.S., single, was treated by me for a chronic gonococcal urethritis and a subacute cervicitis, also gonococcal. After numerous tests of cure, including a complement fixation test, she was discharged as cured. She went to New Zealand, and from there I received a pathetic letter stating that the disease had recurred. Now I had never been more confident of cure than in this patient, and replied to this effect. She returned to me a few weeks later and still complained of an irritating discharge. She also stated that she had seen three gynæcologists in New Zealand, each of whom suspected a relapse, but none could find gonococci in smears. She had been given various treatment. On examination I found trichomonas in the discharge, but no evidence of any recurrence of her former condition. The condition rapidly cleared under treatment.

I have also treated a case of trichomonas vaginitis which developed after I had produced an artificial menopause with the intrauterine application of radium for a non-malignant condition in a patient aged forty.

Apart from the fact that the trichomonas rapidly loses its motility and dies when subjected to temperatures below body heat, it is also very easily killed, both *in vivo* and *in vitro*, by a very wide range of chemicals, which include antiseptics, dyes, lactic acid *et cetera*. Davis and Colwell[4] reported the action of these, using as a control distilled water, in which activity was retained for fifteen minutes. Some of their results are:

Substance.	Strength of Solution.	Result.
Mercurochrome ..	1%	No motility after 3 minutes
	5%	No motility after 15 seconds
Glycerine	Full strength	Immediate loss of motion
Lactic acid	0·5%	No motility after 3 minutes 40 seconds
	2%	Motility ceases after 45 seconds
Methylene blue ..	1%	Slight motility after 15 seconds
Liniment of soft soap	100%, 50%, 10% 1%	Kill instantly Organisms disappear in 30 seconds

Some of these solutions have been used in the treatment, which theoretically should not present any undue difficulty, but which practically is quite a different matter.

Clinically the condition is met with in females at all ages; in both virgins and married women, non-pregnant and pregnant. I have not seen a case before puberty, but Cornell, Goodman and Matthies[5] report their knowledge of such a case in a female child of three years. It is possible that it is really more common in female children and that many cases of vulvo-vaginitis labelled gonococcal may have been of this nature. In America it is reported to be frequently present during pregnancy and may possibly be associated with or even responsible for puerperal morbidity.

Signs and Symptoms.

The signs and symptoms are usually so definite as to form a syndrome almost specific. The outstanding symptom is, of course, a vaginal discharge, which in consistence is thin, and in colour usually yellow, but varying from a very pale yellow to a dark greeny yellow. It is often frothy, containing what appear to be very small bubbles. It is very offensive; indeed it has an almost characteristic odour with a peculiar pungency. It is strongly acid in its reaction to litmus. Irritation is almost always present and causes such symptoms as: (i) *pruritus vulvæ*, (ii) dysuria, (iii) a peculiar burning sensation in the vulva, (iv) intertrigo, (v) dyspareunia. As a result of both the obstinacy of the condition and the ineffectiveness of treatment, the patient is often depressed and neurasthenic.

The signs are of equal importance. Apart from the discharge and odour, there can often be seen on the inner surfaces of the *labia minora* and on the vestibule, small circumscribed areas of a dark red or injected appearance which do not bleed, but are sensitive and tender to touch. When a vaginal speculum is introduced one often notices a thickened, somewhat "granular" condition of the vaginal mucosa, which is bathed with pus and bleeds slightly after swabbing. The appearance is very similar to that of the gonorrhœal granular vaginitis of pregnancy.

The cervix very often has a most characteristic appearance, usually present in severe cases and when no treatment has been previously given. The mucous membrane, particularly around the external os, has a peculiar "strawberry" appearance which was first described by Kleegman. This area also bleeds when the discharge, which is not tenacious, is swabbed away.

The discharge tends to collect in the *cul-de-sac* of the posterior fornix, and this collection is liable to be left behind after treatment if the speculum does not completely "iron out" the posterior vaginal wall. The discharge obtained from this site is the most suitable for diagnosis, for in it the organisms are more numerous and more motile than elsewhere. The cervical canal is quite unaffected. The discharge is particularly profuse following menstruation, after which the condition often seems to recur when previously considered to be cured.

Diagnosis.

The diagnosis can easily be made by microscopic examination of a few drops of fresh discharge under a cover slip. The discharge should be obtained from the posterior fornix, rapidly transferred to a warm slide, and diluted with an equal volume of warmed normal saline solution.

The trichomonas is rounded, oval, or pear-shaped, and varies in size, being sometimes a little smaller

than an epithelial cell and at other times very little larger than a pus cell. The latter type is the most difficult to distinguish, but all forms are easily recognized by their motility, and particularly by a characteristic oscillating or darting movement, when they appear to be savagely attacking the pus cells. All motility quickly ceases in the cold, and in order to obtain a satisfactory diagnosis the conditions above outlined must be strictly observed.

Mention must be made of the fact that gonococci are rarely, if ever, present in vaginal smears obtained from a patient with gonococcal urethritis or cervicitis, unless there be also present a "granular" vaginitis, already referred to, or else in the case of gonorrhœal vulvo-vaginitis of children. Adults rarely develop gonorrhœal vaginitis.

Treatment.

In spite of the susceptibility of the trichomonas to a very wide range of antiseptics of various strengths, the condition remains most intractable to almost all forms of treatment. The tendency to relapse is particularly disappointing and the condition cannot be regarded as cured until the trichomonas is no longer present in the vagina. The best treatment has been found to be that in which the perineum, vulva and vagina are first cleansed with ether soap, and then treated by the topical application of such antiseptics as mercurochrome, methylene blue, brilliant green and glycerine, or by such substances as kaolin or an alkaline powder. Kleegman's treatment consists of the application of mercurochrome and Lassar's paste. Sure and Bercey[6] recently have reported promising results using pulverized quinine sulphate.

Ratcliffe, quoted by Greenhill, experimenting with rats which harbour trichomonas in their intestines, was able to produce a rapid change in the intestinal flora by feeding them with a form of resorcinol, certain types of trichomonas rapidly disappearing. Others have used this as a rational basis of treatment. Using a similar preparation, I have now treated a sufficient number of cases, obtaining such immediate improvement and such rapid cure that it would appear almost specific in its efficacy. The preparation I use is one marketed by British Drug Houses as "S.T.37", which is an aqueous solution of hexyl resorcinol with glycerine added. The solution is cheap, but its great asset is its colourless appearance which is of very great comfort and economical benefit to the patient.

The technique has been as follows: A patient receives treatment on three successive days, then each alternate day for three more treatments. On those days when no treatment is given, the patient takes a vaginal douche of lactic acid, one fluid drachm to a pint. This is continued until the next menstruation, after which any discharge is examined. In many cases this has sufficed for a cure, but in some trichomonas was still present, although there was very little discharge. These patients received a second course of treatment and cure was obtained. In one hospital patient with a very severe discharge, six treatments only, at intervals of one week, *plus* the use of the lactic acid douche, resulted in cure. In every case the first application of the above treatment gave immediate relief. For the actual method great care must be taken to enable the solution to reach all folds and rugæ, after very careful drying of the mucosa following the application of green soap. For the douche I insist on a douche can and glass nozzle, the patient being told to boil the latter before use. All vulcanite nozzles cannot be too strongly condemned. Finally, patients are tactfully instructed regarding their toilet, and are told that the sweep of the arm should be away from the vagina.

In conclusion, it must be fully realized that the conditions under which this pathological condition develops are as yet undefined and must remain so until the life history of the *Trichomonas vaginalis* has been completely described. Then prophylaxis will be understood and curative treatment rationalized. The condition must always greatly interest any practitioner, general or specialist, to whom a female patient presents herself complaining of a vaginal discharge and suffering much mental distress. But only through accurate diagnosis of every such case can treatment prove effective and speedy relief eventuate.

References.

(1) J. P. Greenhill: "Treatment of Trichomonas Vaginalis Vaginitis", *The Journal of the American Medical Association*, Volume XCVI, May, 1931, page 1862.
(2) Ratcliffe: Quoted by Greenhill.(1)
(3) I. F. Stein and E. J. Cope: *American Journal of Obstetrics and Gynecology*, Volume XXIV, September, 1932, page 348.
(4) C. H. Davis and C. Colwell: "Trichomonas Vaginalis, Donné: Preliminary Report on Experimental and Clinical Study", *The Journal of the American Medical Association*, Volume XCII, January 26, 1929, page 306.
(5) E. L. Cornell, L. J. Goodman and M. M. Matthies: "Culture, Incidence and Treatment of Trichomonas Vaginalis", *American Journal of Obstetrics and Gynecology*, Volume XXII, September, 1931, page 360.
(6) J. H. Sure and J. E. Bercey: *American Journal of Obstetrics and Gynecology*, Volume XXV, January, 1933, page 136.

Reports of Cases.

FATAL POISONING BY SODIUM NITRITE.

By ARTHUR A. PALMER, M.B., C.M., F.R.C.S. (Edinburgh),
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A man, aged thirty-nine years, was seen to be in a bad way in his own back yard, about 10 a.m. He exclaimed to a neighbour: "I have just taken something: I thought it was epsom salts, but whatever it was it has got me." He then lay on the ground and writhed about, apparently in pain. Dr. J. J. McIntosh was sent for, and on arrival about 10.30 a.m. found the patient unconscious, deeply cyanosed and sweating profusely. The pulse was slow, the respiration slow and weak, the legs rigid, the pupils contracted. In spite of treatment the patient died at 11 a.m., without regaining consciousness.

Dr. C. B. Howse, who carried out the autopsy, found nothing characteristic of any poisoning.

Analysis by Mr. S. G. Walton, the Government Analyst, showed that the supposed salts was nitrite of soda, and a considerable quantity of this was present in the stomach.

It was given in evidence before the Coroner that the sodium nitrite had been brought from the abattoirs, where it was used to maintain the fresh appearance of meat. The deceased's widow stated that she used it in place of saltpetre for pickling meat.

This case is recorded as fatal poisoning by the nitrites appears to be very rare.

Reviews.

AMORALITY.

"THE present desperate plight of our civilization" has led to the suggestion of many peculiar remedies. The latest comes from the pen of R. E. Money-Kyrle, and apparently in all seriousness.[1] His book, "Aspasia: The Future of Amorality", reveals him as a misguided enthusiast. To him morality means sexual morality. He has attempted what he calls "a survey of the psycho-analytic theory of social evolution". He has examined the defects in present day culture from the psycho-analytic standpoint and has tried to diagnose their cause. He holds that the Œdipus complex is at present inescapable and that it is not only directly responsible for much of the unrest and depression of the present time, but that it is also a cause of national and social animosity. His remedy is sexual freedom—what he calls Aspasia or amorality. To do him justice, he admits that much of his argument is necessarily speculative. Perhaps he is wise in calling his book Aspasia, the name of "the most celebrated of courtesans". We feel sure that if the author tried he could find reasons, convincing at least to himself, for the removal of all moral control. We are afraid that, though Aspasia "was intelligent and much maligned", we must banish her.

CHRONIC RHEUMATISM.

J. D. HINDLEY-SMITH, in his book entitled "Chronic Rheumatism and the Pre-Rheumatic State", hopes to demonstrate the clinical grounds upon which he makes a number of interesting assumptions.[2] He assumes that:

> (1) Chronic rheumatism is a terminal symptom arising from a toxæmic condition of considerable duration. (2) The toxin in question always possesses similar pathological effects, although a variety of causes may give rise to it and although the toxin itself may vary somewhat in constitution. (3) The pathological effects, among them being chronic rheumatism, are in all probability a resultant of the interaction of the toxin and the physiological resistance of the body, and are not the direct effect of destructive action upon the affected tissue by the toxin. (4) The degree of interaction which occurs in the body in response to the toxin depends to a large extent upon "allergic sensitivity". Sensitivity may be hereditary or, more rarely, acquired. (5) The direct cause of the toxin may be either infective or metabolic, the percentage being approximately 95% infective and 5% metabolic. (6) Of the above causes, the best known metabolic disorder which results in chronic rheumatism is gout. Of the chronic infections producing chronic rheumatism approximately 25% of cases are caused by dysentery, gonorrhœa and certain pathological intestinal bacteria, and approximately 75% by a certain streptococcal infection.

He concentrates his attention upon this streptococcal group and suggests the name "chronic streptococcal toxæmia" for the disease it produces, regarding the term "rheumatism" as applicable only to a symptom common to many diseases. The primary stage of the disease is usually demonstrable at an age as early as four or five years, "as a general streptococcal infection of the naso-pharyngeal mucosa, causing a localized catarrhal condition and, commonly, an inflammatory turgidity of the tonsils and a deposit of adenoid tissue. Between the ages of approximately nine and twelve years the condition becomes less local in character, with constitutional signs, for example, growing pains, anæmia, acidosis *et cetera*. This is the onset of the secondary stage characterized by constitutional debility which extends through adolescence up to middle life. At this time, approximately the age of forty-two, the tertiary stage begins, the chief characteristic of which is chronic rheumatism in all or any of the forms in which this complaint is recognized".

The early diagnosis and appropriate treatment before any harm has occurred to the patient should be aimed at to check a condition which, apart from the sufferings of the victims, is responsible for the incapacity for work of fully one-sixth of the working population of the United Kingdom at a cost of two million pounds a year for sick benefit.

We regret that the author is somewhat vague about the cultural methods adopted for the diagnostic throat and nasal swabs, but pathogen-selective culture should prove of distinct value.

The prolific and energetic measures advocated in the treatment should stimulate practitioners who read this very readable and inexpensive little book.

FRACTURES.

A TREATISE on fractures often presents itself as a volume of well over a thousand pages. This one, entitled "Treatment of Fractures in General Practice", by W. H. Ogilvie, contains 180 pages in all, distributed over two volumes of pocket size, and the English published price of each volume is only half a crown.[1] It is a very good money's worth. Small as it is, it is a most successful practical treatise, the work of an experienced man who knows where the trouble is going to be, and tells his readers in plain, easily understood language what he thinks they should do. It may be that here and there we would advocate some other way, but we cannot expect always to agree about everything, and at all events the methods given in the book are supported by respectable authority. We find the teaching of the late Robert Jones followed as regards the use of certain types of splint, but we can also see that Böhler has had a great influence on the author's practice and teaching. He has, for instance, followed Böhler in advocating local anæsthesia for reduction of practically all fractures. This is the first time we have found this excellent improvement in fracture treatment incorporated in a text book other than Böhler's own book. This, however, is only one of the signs of Böhler's influence. There are many others, and perhaps in one or two cases we feel we should like to see a method tried on somebody else before submitting to it ourselves. We note that the author considers the habit of calling all malleolar fractures "Pott's fracture", a foolish one, and he compares it with the similarly foolish and confusing French habit of calling all malleolar fractures "Dupuytren's fracture". He is quite right. Exactly the same thing was said long ago in THE MEDICAL JOURNAL OF AUSTRALIA.

Let us conclude by saying that although this book is such a little one, it is one of the best practical guides on fractures.

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[1] "Aspasia: The Future of Amorality", by R. E. Money-Kyrle, M.A., Ph.D., with introduction by J. C. Flugel; 1932. London: Kegan Paul, Trench, Trubner and Company, Limited. Crown 8vo., pp. 141. Price: 5s. net.

[2] "Chronic Rheumatism and the Pre-Rheumatic State", by J. D. Hindley-Smith, M.A., M.R.C.S., L.R.C.P.; 1932. London: H. K. Lewis and Company, Limited. Crown 8vo., pp. 154. Price: 5s. 6d. net.

[1] "Pocket Monographs on Practical Medicine: Treatment of Fractures in General Practice", Volumes I and II, by W. H. Ogilvie; 1932. London: John Bale, Sons and Danielsson, Limited. Foolscap 8vo., pp. 180, with illustrations. Price: 2s. 6d. each volume.

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HOSPITALS AND THEIR EQUIPMENT.

WITH the advance in medical science during recent years there has been a corresponding change in the type of investigation and treatment undertaken at hospitals. Whereas in former years hospitals were little more than nursing homes, they have now become institutions staffed by a multitude of specialists, armed with diverse and complex apparatus and equipment, and stocked with medicaments in the form of special drugs and biological products that were not dreamed of fifty years ago. In bygone days people did not often seek a hospital bed of their own accord. Admission to hospital was something to be feared. If their homes were suitable, people elected to be nursed at home, and as a general rule they fared just as well at home as in the ward of a hospital. Nowadays people know that diagnosis and treatment often require the horde of specialists, the wealth of equipment and the store of medicaments. They face with equanimity, if not with cheerfulness, the prospect of occuping a hospital bed. In this way what is known as the hospital problem has arisen. People for whom the public hospitals were never intended are content, even anxious, to get something for nothing. And it is true that the public hospital patient, at least in Australia, receives more efficient treatment than any other class of patient, because he has the advantage, should he require it, of the collaboration of many medical attendants.

The functions of hospitals are many. They need not be discussed or even enumerated at the moment. Reference will be made to one aspect of the main function only, the adequate treatment of the sick. If a patient is to receive adequate treatment, he must be able to command every method of investigation that may throw light on his condition, and he must have at his disposal every modern method of treatment. In most of the larger metropolitan hospitals in Australia and in some of the country hospitals all these things are possible. It is unfortunately true, however, that in many places not only are modern facilities in equipment and so forth not available, but there is a stolid and stupid complacency that inhibits any effort to provide them. The most obvious deficiency is generally seen in regard to pathological work. In some large country centres the simplest pathological and biochemical equipment is lacking. Even the capital cities are not beyond reproach. For example, at Hobart, the capital city of a State, the General Hospital has no pathologist on its staff. In some instances the medical practitioners attached to the institutions are to blame, for they do not insist on a high standard. Sometimes the recommendations of the staff are ignored.

What is to be done? Australia is not alone in this problem; most countries share it. In America an attempt has been made to improve the efficiency of hospital work by standardizing or grading the hospitals into certain classes. Though some critics would have us believe that the results of the scheme look well on paper and no more, we do not believe that these critics are right. A grading of hospitals, based *inter alia* on the provision of apparatus and equipment, would be an advance for Australia. Once the facilities were available, the lazy members of the medical profession would at the worst be shamed into using them. If any move in this direction were made, it should emanate from the

profession as a whole. It would need to be made by a body with an organization extending throughout the Commonwealth. The only two bodies so qualified are the Federal Council of the British Medical Association and the Royal Australasian College of Surgeons. The organization of the Federal Council is scarcely complete. On the other hand, if the Royal Australasian College of Surgeons initiated the grading of hospitals, it would give a wrong impression, an impression only too prevalent, that hospitals are chiefly places at which surgical operations are performed. The Federal Council should consider the wisdom of such an undertaking. Having formulated a plan, it might find ways and means of carrying it out. Moreover it might invoke the aid of the Royal Australasian College of Surgeons or even prevail on the College to undertake the work; the scheme would in these circumstances have the backing of the whole profession. At the present time a subcommittee of the Federal Council is investigating the hospital problem. Hospital grading might well be considered as an addendum or as complementary to the report the subcommittee will make.

Current Comment.

PYRIDIUM AND SERENIUM AS URINARY ANTISEPTICS.

THERE is no absolute proof that any known drug taken internally acts as an efficient urinary disinfectant. Disinfection of the urinary tract presents exceedingly complicated problems. Before a drug administered orally can act as a urinary disinfectant it must be absorbed from the gastro-intestinal canal, pass through the body and be excreted by the kidneys. Most antiseptic substances given by mouth lose their antiseptic properties before being excreted in the urine. To find a drug which will be excreted in the urine in sufficient concentration to sterilize infected urinary passages presents a problem which has not yet been solved. The ideal urinary antiseptic must be non-toxic in therapeutic doses. It should be chemically stable; it must not irritate the urinary passages. It should be excreted in a high percentage in the urine and should exert its antiseptic effect in high dilution in urine either acid, alkaline or neutral, and its antiseptic activity must be continuous. Acriflavine and proflavine, according to E. G. Davis, are antiseptic in doses of five milligrammes per kilogram to rabbits whose urine is normally alkaline, but not to dogs with acid urine. Proflavine and acriflavine, given orally in doses of one gramme, are said to be excreted in the urine of normal persons in sufficient concentration to render the urine, provided it be alkaline, an unfit culture medium for the colon bacillus and staphylococci. The antiseptic action becomes evident about two hours after administration and lasts for eight hours. Intravenous administration is more prompt and efficacious in smaller doses. The toxic limits have not yet been accurately ascertained. These investigations need confirmation.

Two complex azodyes of the pyridine series, extolled as urinary antiseptics, efficient both in alkaline and acid urine, are neotropin and pyridium. They are both advocated for cystitis, pyelitis, pyelonephritis and gonorrhœal infections. Both are stated to be very penetrative. They are eliminated partly by the liver. Pyridium contains 28% of nitrogen. Both are non-toxic and non-irritant, readily absorbed and rapidly excreted through the urinary tract.

J. B. Gillespie has experimented on the antibacterial properties of pyridium and another reputed urinary antiseptic, serenium.[1] In regard to pyridium, he observes that the only toxic effects recorded are nausea and vomiting in children. Contraindications are stated to be uræmia, chronic parenchymatous nephritis and non-infective disorders of the kidney and severe hepatitis. Pyridium should not be given with mercury compounds, as it releases the mercury. Being relatively insoluble, some is lost in bowel evacuations. Aqueous solutions are highly acid. For estimating bactericidal activity *Escherichia communior* and *Staphylococcus aureus* were used. Both grew vigorously for seventy-two hours in sterile distilled water of a pH of 6·4. This was used in all dilutions and suspensions. A pH of from 4·6 to 5·0 inhibits *Escherichia communior* from growing in urine. Therefore, buffered controls having a pH corresponding to that of the solutions to be tested were used. These buffered controls were made from solutions of sodium phosphate and citric acid. Gillespie found that approximately 50% of pyridium administered by mouth is excreted in the urine within twenty-four hours. Smaller amounts are eliminated in the urine for several hours later. When 0·8 to 1·0 gramme is given daily, and fluid intake is restricted, a concentration in the urine of one in 1,600 to one in 2,400 may be attained. Gillespie points out that experiments *in vitro* indicate that the bactericidal action of pyridium in aqueous solution is greater against colon bacilli than against *Staphylococcus aureus*. Solutions of one in 1,000 to one in 7,000 destroy colon bacilli within four hours. Except the one in 1,000, such concentrations are ineffectual against staphylococcus in from twenty-four to forty-eight hours. The addition of urine to aqueous solutions of the drug markedly reduces bactericidal activity. The destruction of bactericidal ability is directly propor-

[1] *American Journal of Diseases of Children*, February, 1933.

tional to the amount of urine present. After oral administration, urine containing the excreted drug in concentrations of one in 1,600 to one in 1,800 has only slight bacteriostatic and no bactericidal effect on *Staphylococcus aureus*. Gillespie finds that in such concentrations *Escherichia communior* is neither killed nor is its growth inhibited. Normal blood serum also reduces the antibacterial properties of pyridium *in vitro*. There is evidence that pyridium given by mouth is altered in its passage through the body and that the dye excreted in the urine is not pyridium.

Serenium is an organic dye of different composition, said to be relatively non-toxic and excreted almost entirely in the urine, to which it imparts an orange or red colour. Gillespie found it to be very insoluble and the amount excreted in the urine to be much less than that ingested. Probably much is eliminated by the bowel. With restriction of fluids, a concentration of one in 4,000 was obtained, but with moderate restriction of fluids and using the doses recommended, concentrations varied from one in 10,000 to one in 16,000 or even higher dilutions. Serenium in aqueous solutions of one in 6,000 to one in 8,000 is bactericidal for Escherichia. Solutions of one in 6,000 to one in 8,000 are bacteriostatic, but not bactericidal for *Staphylococcus aureus*. When urine was used in making dilutions the antibacterial properties were lost as regards Escherichia. A change in the appearance of the urine-containing solutions on standing and the complete loss of antibacterial properties suggest to Gillespie a chemical reaction between the drug and some urinary constituent. Serenium causes no such change; so probably the drug is chemically altered in its passage through the body. Serenium excreted in the urine in concentrations of one in 4,000 to one in 8,000 or even one in 16,000 is bacteriostatic for staphylococcus. A concentration of one in 4,000 is slightly bacteriostatic for Escherichia; one of one in 8,000 is not. No bactericidal effect on either organism was noted.

Antisepsis in urine is quite different from antisepsis in water. Drugs highly bactericidal in water may completely lose this power in urine. The value of so-called urinary antiseptics must be questioned until the antiseptic strength can be experimentally demonstrated in urine. Compounds, the antiseptic power of which is due to acid or basic properties, would become inert in urine owing to the buffer action of urinary salts. Compounds containing silver become inert in urine owing to precipitation of silver chloride. Some unknown interfering action may cripple the antiseptic effects of certain compounds, and this probably happens with pyridium and serenium. The urinary constituents may chemically alter such compounds. The antibacterial action of pyridium and serenium is quite independent of the Gram-staining quality of the organisms tested. In watery solutions both were more effective against Escherichia than against staphylococcus. The excreted drugs, however, had an inhibitory action against the Gram-positive organisms, while Escherichia grew well. The antibacterial powers of the drugs are profoundly influenced by the media in which they act. Both are eliminated in the urine in amount sufficient to cause discoloration of that fluid. This adds another difficulty to the complexities of the situation. The conspicuous staining of the underwear and night attire due to such drugs as pyridium, neotropin and serenium is most objectionable to sensitive patients who do not wish their infirmity proclaimed abroad. Although one of the dyes may yet prove efficacious as an antiseptic, æsthetic considerations may prevent its general use.

MULTIPLE PRIMARY MALIGNANT GROWTHS.

THE fact that more than one primary malignant growth may occur in the same person is well known. The occurrence, however, is held to be rare. Ewing points out that tumours have been observed simultaneously in the uterus, ovaries and breast, and adds that this combination suggests the influence of the functional relation between these organs. He also states that the occurrence of two or more tumours in different organs of the same subject suggests nothing more than the accidental coincidence in several organs of the general ætiological factors in the genesis of tumours. Ewing refers to Hausemann's observation of five multiple primary tumours in one thousand autopsies in cases of tumour, and to the 1,225 cases of tumour, collected by Redlich, which included 14 with more than one tumour. Bland Sutton states that the occurrence of two primary cancers in the same patient is excessively rare. H. H. Hurt and A. C. Broders, in a recent report from the Mayo Clinic, show that persons are much more likely to have more than one primary malignant neoplasm than the literature would lead one to believe.[1] Of the 2,124 patients with malignant neoplasm seen at the Mayo Clinic in 1929, 3·4% had more than one primary malignant neoplasm. They give sound reasons for assuming that the real percentage was higher than 3·4, but these need not be considered at present. In their investigations these two authors used the well known Broders method of grading tumours. There were 150 lesions in 71 patients. It was found that the average grade of malignancy varied directly with the number of lesions in any one age group. This is explained by the statement that if a person has one malignant tumour, the probability of the development of another malignant tumour varies directly with the grade of malignancy of the first tumour, presuming that the person survives long enough for another malignant newgrowth to develop. Though they do not state it definitely, the authors appear to lean to the view that the factors causing a single newgrowth are exaggerated in cases of multiple primary malignant newgrowths. This hypothesis would be worthy of investigation by means of the Broders method of grading tumours.

[1] *The Journal of Laboratory and Clinical Medicine*, May, 1933.

Abstracts from Current Medical Literature.

MEDICINE.

Poisonous Spider Bites.

E. BOGEN (*Annals of Internal Medicine*, September, 1932) describes the symptoms and treatment of bite by the *Latrodectus mactans*, a poisonous spider, commonly called the black widow or shoebutton spider, which is the cause of most poisoning symptoms in the United States of America. The spider is black with red markings on the abdomen. As a rule patients are bitten in an outdoor privy or a garage, but sometimes in bed. The bites are on the penis or near the penis in half the cases, but the extremities, body and head are also the site of bites. Severe pain, spreading from the bite all over the body, and especially affecting the abdomen, with rigidity of muscles is usual, but tenderness is slight. Increased blood pressure, active reflexes, a slight rise of temperature and leucocytosis are noted, with restlessness, sweating, nausea and vomiting, retention of urine and spasm or twitching of muscles. Increased intraspinal pressure is noted. Lumbar puncture gives great relief from symptoms. Convalescent serum, from two to thirty-five cubic centimetres injected intramuscularly, if given within eight hours, gives relief. Local application of tincture of iodine, hot baths or hot compresses and opiates or hypnotic drugs are advised. Various acute conditions, such as appendicitis, renal colic and other abdominal conditions have been diagnosed in these cases and operations have been performed needlessly. Incision, scarification and local antidotes are useless and possibly harmful. Recovery is the rule, though a number of fatalities have been reported. The author likens this spider to the Australian *Latrodectus hasseltii*, and quotes reports in THE MEDICAL JOURNAL OF AUSTRALIA on this subject.

Calcium Metabolism.

A. R. BERNHEIM (*The Journal of the American Medical Association*, April 1, 1933) discusses calcium need and calcium utilization. Sherman states that 0·45 gramme of calcium is the minimum and 0·70 gramme of calcium (1·0 gramme of calcium oxide) is the optimum daily requirement for adults. He showed the beneficial effect in rats of increased calcium through an additional amount of milk; better health and longer life were the result; weakness and infection were lessened. Human diet is often deficient in calcium, and it may be that the calcium depletion of bones in old age and other manifestations of ill health are the result of prolonged calcium defect in the diet. Milk and cheese are the only substances which give a reasonable supply of calcium in comparison with their weight; for instance, 0·675 gramme (one and a half pounds) of milk or 0·11 gramme (one-quarter pound) of cheese yields 0·70 gramme of calcium, whereas it requires 11·7 grammes (twenty-six pounds) of lean meat or 3·2 grammes (seven and three-tenths pounds) of white bread to yield the same quantity. Calcium is absorbed through the small intestine; when the medium is alkaline, insoluble calcium salts are formed and absorption is hindered; measures which promote intestinal acidity favour absorption. Fats hinder absorption, as does oxalic acid, present in leafy vegetables. Oxalic acid combines with calcium, forming insoluble calcium oxalate, which cannot be absorbed. Lactose favours absorption, possibly due to the formation of lactic acid in the intestine; other sugars and carbohydrates hinder absorption. Orange and tomato juices, by their content of vitamins *A*, *B* and *C*, and of citric acid, along with a high calcium diet and vitamin *D*, promote calcium absorption. Vitamin *D* especially has this function, and it is suggested that where adequate exposure to sunlight is unobtainable, vitamin *D* in the form of concentrated cod liver oil or viosterol should be given three or four days a week. In a recent report Cantarow considers the use of calcium salts in tetany, jaundice, hæmorrhage, œdema, lead poisoning, tuberculosis, ulcerative and mucous colitis.

The Fat-Soluble Vitamins.

E. MELLANBY (*Edinburgh Medical Journal*, April, 1933), in a review of the fat-soluble vitamins, points out that though vitamin *D* is the master key to bone calcification, there are other constituents of food which influence bone calcification and growth, and that however much vitamin *D* is ingested, bone deficit must result if the diet is very deficient in calcium or phosphorus. Cereals are rickets-producing substances, partly because they lead to increased growth without at the same time supplying to bones a sufficiency of calcium, phosphorus and calcifying vitamin, and partly because the calcium and phosphorus they contain are not retained in the body. The latter occurrence is especially true of those cereals which are relatively rich in calcium and phosphorus, such as oatmeal and wheat germ. In order to retain the good and eliminate the harmful qualities of cereals it is essential to give them with foods rich in calcium and phosphorus, such as abundant milk, and to increase the vitamin *D* intake. Calcium appears both to antagonize the anticalcifying effect of cereals and to aid the action of any vitamin *D* present in the diet. It has not yet been found possible to establish any direct relationship between vitamin *D* and either phosphatase or the parathyreoids or the endocrine function, and a large field of investigation remains for their correlation. Both fat-soluble vitamins stimulate growth in young animals. The earlier work on fat-soluble vitamin deficiency, interpreted as being due to absence of vitamin *A*, was really the result of a deficiency of both vitamins. The present state of knowledge of the action of vitamin *A* is far less advanced than that relating to vitamin *D*. It is probable that, as with vitamin *D*, the action of vitamin *A* is not a simple story, and that it will ultimately be found that it works in harmony with some dietetic factor to promote resistance of mucous membranes and epithelial cells to micro-organisms, while other factors, such as cereals, antagonize its influence. Vitamin *A* has been found of value prophylactically and therapeutically in certain human infections. A deficiency of vitamin *A* only will produce demyelination and ultimately disappearance of nerve fibres in the anterior columns, the cerebellar tracts and the posterior columns of the spinal cord and in the afferent fibres of other peripheral nerves, such as the optic, the vestibular, cochlear and trigeminal nerves. These changes are made worse if wheat embryo be substituted for some of the white flour in the dietary. The symptoms are suggestive of convulsive ergotism, and a few grammes of ergot added to the dietary will increase the degenerative changes, but if an abundance of substances containing vitamin A be given, ergot has no effect on the central nervous system. Epidemic ergotism is due not so much to the consumption of ergotized rye as to the lack of vitamin *A* due to drought and famine. Yellow maize contains carotine and pellagra is found where white maize but never where yellow maize is eaten. The skin changes of pellagra may be lesions of a trophic nature related to nerve degeneration, and not to deficiency of vitamin B_2, as has been thought. Lathyrism may have a similar ætiology. The fact that vitamin *A* and probably other dietetic factors increase the resistance of nervous tissues to those toxins which tend to produce demyelination, while cereals tend to favour such degenerative changes, should be taken into account in the consideration of disseminated sclerosis, subacute combined degeneration of the cord and the manifestations of neurosyphilis.

Treatment of Œdema of Nephrosis.

A. F. HARTMAN, M. J. E. SENN, M. V. NELSON AND A. M. PERLEY (*The Journal of the American Medical Association*, January 28, 1933) have recorded some results of treatment of œdema by intravenous injection of acacia. In the persistent œdema of nephrosis, unrelieved by high protein and salt-free diet, intravenous injection of one to two grammes of acacia per kilogram body weight (30% acacia diluted with equal parts of Ringer's solution if the original preparation is salt-free, or diluted with equal parts distilled water when the acacia solution contains sodium chloride) caused diuresis and diminution or loss of œdema in many patients. As many as eight such injections were given at intervals of two days to three weeks. Six patients were treated; all had been

given diuretics and had received other recognized means of treatment before receiving acacia injections. The effect of the acacia is to raise the colloidal osmotic pressure or oncotic pressure of the blood serum. Apart from relief from œdema, general improvement was observed in two patients and temporary benefit in two others.

The Use of Bacteriophage in Bacteriæmia.

W. J. MACNEAL (*The American Journal of the Medical Sciences*, December, 1932) discusses bacteriophages and their clinical applications in the treatment of bacterial infections. The treatment of infectious disease by the use of bacteriophage presents problems depending on the location of the infectious process. Where the blood stream is infected, one is practically always dealing with a pure culture of bacteria. A special filtrate, prepared in a culture medium without protein and containing asparagin as the important source of nitrogen, is injected intravenously. This preparation does not of itself produce serious toxic symptoms in animals, and it seems to be far less dangerous to the patient than is the filtrate of broth culture. Some instances of recovery under this treatment have been observed in cases of staphylococcus bacteriæmia after repeated positive blood cultures.

Lead Poisoning and Gastric Ulcer.

A. PEIPERS (*Deutsche Medizinische Wochenschrift*, February 17, 1933) quotes Schiff to the effect that, apart from the typical lead colic, uncharacteristic long lasting pain occurs in lead poisoning. This is often associated with a hyperacidity and a true duodenal ulcer. Peipers asks: Is there a relationship between duodenal ulcer and chronic lead poisoning? In the causation of ulcer two chief factors must be considered: (i) a disturbance of the circulation due to some cause; (ii) a loss of balance between the peptic activity of the stomach juice and the antipeptic protection of the stomach wall. Circulation disturbances in the form of vessel spasm can occur and there may also be muscular hypertrophic thickening of the connective tissue of the blood vessel spaces and obliteration of the vessels. Owing to the frequency of ulcers, one could conclude that the occurrence of both together was accidental. In the author's case he thought it justifiable to conclude that the chronic lead intoxication was the cause of the ulcers since the symptoms occurred only when symptoms of lead intoxication appeared clinically. Several other cases of this relationship have been described in the literature.

Appendicitis.

A. KRECKE (*Münchener Medizinische Wochenschrift*, February 24, 1933) writes that the true cause of acute appendicitis is still unknown. One must assume that it is due to an organism acting on the lumen of the appendix, especially streptococci or pneumococci. Certain mechanical causes play a part in the disease. Neuro-angiospastic disturbances must be considered in those cases in which the disease attacks several members of the one family. An occasional endemic occurrence of the disease must be admitted. The effects of nutrition cannot generally be blamed, whilst trauma may be only very rarely recognized as a cause of appendicitis.

Pernicious Anæmia.

H. SCHEIDEL (*Münchener Medizinische Wochenschrift*, February 24, 1933) writes that 269 cases of proven pernicious anæmia were observed in the medical clinic of Heidelberg during the past twenty and a half years. The disease was more common in females and has increased in frequency since 1913. Before the war the number was 0·13% of the patients admitted, and after the war 0·36%. It was most frequently observed between the fiftieth and fifty-ninth year. The blood picture is the most deciding factor in diagnosis. Rarely do hypochromic cases occur, and achylia is always present, except in very rare instances. The following changes were noted: elevation of temperature in 52%, loss of weight in 47%, frequent clinical and X ray heart changes, hypotonia in 26%, changes of the tongue in 50% of the cases. The spleen was enlarged in 75% of the cases; urobilinogen and urobilin were increased in the urine in 80% of the cases. The sedimentation rate was increased in 75%. The majority of the patients were of the asthenic rather than of the sthenic type. Nervous changes increased in frequency in the last few years, and this was especially noticeable soon after the general usage of liver therapy. No opinion can be given of the duration of the disease, since the actual onset cannot be determined. Liver therapy has certainly prolonged the life of the patient. Before the introduction of liver therapy, the average duration of life was only one to one and a half years. Forty-six out of the 269 patients died (40 up to 1927, and six from 1927 to 1933). Eight patients had anæmia of the acutely progressive type and died in periods varying from five weeks to four months.

The Depôt Treatment of Pernicious Anæmia.

L. NORPOTH (*Münchener Medizinische Wochenschrift*, March 17, 1933) has observed that daily injections of "Campolon" can be simplified by giving a collective dose (one single injection of 12 cubic centimetres, the effect of which lasts for six days). It will therefore be possible, by giving larger doses at intervals, to replace the missing substance and this dosage may be smaller than the daily necessary dose. In the author's clinic a definite rule for dosage cannot be given, but it appears that once a patient with anæmia of moderate severity has a normal blood picture, 10 cubic centimetres of "Campolon" given once a month intramuscularly should maintain that blood picture. No experience with other liver or stomach preparations was obtained. The chief value of this treatment is the easy administration and the avoidance of daily injections.

Angina Pectoris Diaphragmatica.

L. HOFBAUER (*Münchener Medizinische Wochenschrift*, March 17, 1933) comes to the following conclusions: (i) Disturbances of the diaphragm at rest and in motion often produce the clinical picture of *angina pectoris*. (ii) Owing to the occurrence of these attacks, there must be some relationship between anginal attacks and a pathological condition of the diaphragm. (iii) If there is any mechanical interference with the movements of the diaphragm (pleural adhesions, increased intraabdominal pressure, diaphragmatic hernia), anginal symptoms will occur, which disappear on removal of the cause. (iv) If the diaphragm is organically sound, but there are changes in the central circulation (coronary artery, myocardium, aorta), a hyperæsthesia of the diaphragm may occur by a viscero-sensory reflex causing difficulty in breathing, painful contraction in the chest, and thoracic restriction. (v) There is a misleading similarity between organic and reflex dysfunction of the diaphragm. In both the end result is hyperæsthesia of the diaphragm causing an arrest in breathing followed by interference with the circulation.

The Corpus Restiform Syndrome (Mann).

K. MENDEL (*Deutsche Medizinische Wochenschrift*, April 7, 1933) records a case of corpus restiform syndrome following a cerebral contusion. This lesion has often been mistaken for a post-traumatic functional lesion. The case was that of a man who fell on to the back of his head and suffered from definite signs of concussion. Since the accident he has complained of left-sided impairment of hearing, noises in the ear, giddiness, vomiting, epileptoid attacks and alteration of habits. The examination revealed slight deficiency in movement of the eyes to the left, the left corneal reflex less active than the right, easier production of left galvanic nystagmus, pass-pointing to the left, deviation to the left on walking, Rombergism to the left and posteriorly, positive left Oppenheim reflex, and hyperæsthesia on the left side. The author concludes that, together with psychic disturbances, there have been organic disturbances, and in view of the symptoms, the localization appears to be in the restiform body. The left Oppenheim reflex is difficult to explain. Mann observed several patients over years without noting any improvement.

Special Articles on Treatment.

(Contributed by request.)

XIII.

THE TREATMENT OF DIABETES MELLITUS.

PART I

IT is quite impossible in a rapid survey to describe in any detail the many facets of the treatment of *diabetes mellitus*. One may say at the outset, therefore, that it is the duty of every practitioner to possess and study a copy of a concise and practical manual such as that provided by R. D. Lawrence or Elliott Joslin. It is practicable here merely to remind readers of a few of the broad principles underlying the present day management of the disease.

It is still common to encounter a number of patients erroneously classed as diabetics and committed thereby to a dreary *régime* of living, and to unremitting anxiety arising from their firm conviction that they suffer from an incurable and eventually fatal disease. Diagnosis must be accurate and decisive at the very beginning of treatment. The renal glycosuric and aspirin addict must be as emphatically reassured as the true diabetic solemnly warned. Once a diabetic, always potentially a diabetic, is an unassailable dictum. The discovery of glycosuria in a person with a family history of the disease must be viewed with grave suspicion. The foreknowledge of such a diathesis should result in the immediate testing of the urine of any such patient, who merely complains of feeling "run down". It is exceptional to find the reagents required for the Rothera test for acetone on the shelves of the general practitioner's surgery, yet this simple determination is invaluable in the rapid discrimination and treatment of the true disorder, as very few diabetics taking an uncontrolled diet fail to show ketosis. Nevertheless, the safest and surest procedure is to insist vehemently that every patient whose urine reduces Fehling's or Benedict's solution, should submit to a sugar tolerance test as soon as possible, regardless of financial circumstances or geographical location. Departure from or delay in enforcement of this rule will sooner or later lead to disaster. With regard to this text: "Remember that a fasting level (of blood sugar) of over 0·13% and a post-prandial level of over 0·2% denote true *diabetes mellitus*" (Lawrence).

With a diagnosis of *diabetes mellitus* established beyond all doubt, what is the primary procedure? The answer is neither immediate starvation nor the administration of insulin, but this: a most complete and searching physical examination, embracing a quest for focal sepsis in teeth, nose, tonsils, ears, prostate, uterine tubes, and especially the gall-bladder and chest. If the patient has even a cough, sore throat or repeated colds, a radiogram of the lungs is essential. Tests should be made of the condition of the peripheral vessels, nervous system, heart, fundi and particularly the ductless glands. By this means, a number of cases diagnosed as *diabetes mellitus*, according to the glucose tolerance curve, will be found to be associated with toxic, endocrine, gestatory or cerebral causes, each requiring special and intrinsic treatment.

The First Diet.

All workers are agreed that the first diet depends entirely on the severity of the accompanying ketosis, rather than on the total amount of sugar passed in twenty-four hours, and on the general condition of the patient. If he is able to walk to the consulting room, has no anorexia, epigastric pain or drowsiness, with the pulse not unduly rapid, nor the eye tension reduced, nor even the tongue sticky or dry, it is as a rule unnecessary to confine him or her to bed, and a commencement may be made with qualitative dietetic restriction alone. A period of preliminary starvation is not only antiquated, but dangerous.

(*a*) Patients in good or fair state of nutrition, who may not give a positive response to the Rothera test in the urine or who may give a response that takes several seconds to develop, and who give no response to the Gerhardt reaction: the majority of mild or semi-treated diabetics fall into this group. They are asked simply to try a qualitative restriction of their carbohydrate intake (and no more). They are given a written list of foods from which they must abstain, and a further list from which they must eat most sparingly. Thus, sugar, fruits, potato, biscuits, jams, honey, treacle, syrups, chocolate, jellies, soft drinks, sweet wines, pastry, cakes, scones, porridge, breakfast foods, thick soups, *et cetera*, are proscribed in their entirety. Bread is allowed up to one and a half slices daily, or as toast, and experience has shown that it is necessary to explain clearly to patients that biscuits, brown bread and toast are every bit as undesirable as white bread, and that commercial diabetic foods are equally to be spurned. If any of the last-mentioned are permitted or afforded, Allenbury's Diabetic Flour seems to be the least noxious. Later on, the educated and reliable patient may be allowed small quantities of some of these foods, up to the point of occasional glycosuria, but in general they must be thenceforward regarded as wholly harmful. Fresh greens are encouraged and effectually prevent any scorbutic tendency. It should be made very plain to the patient that by faithful adherence to this simple *régime* he may be able to evade food weighing and insulin. Benedict's standardized reaction is carefully demonstrated and a written description is provided. The patient is told to keep a chart showing the colours resulting in the early morning urine, together with that passed before dinner and at bedtime. If there is a doubt as to the patient's comprehension or trustworthiness, one must insist on seeing the wife or mother and explaining the details to her. Urinary ketones should be sought for in the early morning urine by the medical attendant himself, every three or four days. This qualitative carbohydrate restriction should be persisted in for at least a fortnight, unless there be reason to proceed more rapidly with stricter treatment by reason of further deterioration in the patient's condition or an increase in urinary ketones. The glycosuria usually diminishes or disappears at once, but if after a fortnight even a trace of acetone is found, the carbohydrates should be gradually increased, up to the point of occasional glycosuria, and if acetone is still present, the patient should be treated as one of the next group. It is well to bear in mind at this juncture that the assessment of the degree or even the presence of ketosis from the odour of the breath alone is most misleading. In the case of the elderly or if the history of the diabetes is a long one, the possibility of a raised renal threshold should now be considered and provided the patient is sugar-free in the early morning, can now be determined by another glucose tolerance test.

(*b*) Patients showing an abundant glycosuria remaining after application of the unweighed diet prescribed above, with or without ketonuria: this class must resign themselves to a temporary but probably permanent abnegation of the pleasures of the table and to the dreary business, at first anyway, of weighing their food. But it is amazing how great the reduction in urinary sugar may be on changing over from a "qualitative" to a "quantitative" diet.

The final caloric value of the diet selected should vary according to the "figure", age and duties of the patient. The first thing to do is to form an approximate estimate of the average daily diet before treatment is begun at all, or the qualitatively restricted diet already being consumed is worked out in calories and grammes of carbohydrate, protein and fat, according to the food values of Lawrence and McCance. No sudden or drastic alterations in diet are ever wise in *diabetes mellitus*, so gradual steps (150-200 calories at a time) are made towards the newly chosen ration.

The fixing and constitution of this new level of food intake (the adjustment of the dietetic carburettor) is just the aspect of diabetes which is disliked and shirked by so many doctors, who in consequence come to rely entirely on "ready-made" schemes such as the "Line

Ration" or "Five Gram" plan, worked out and so successfully applied by R. D. Lawrence. But, apart from considerations of personal incompetence, there is elasticity and variety in a diet prepared by oneself in consultation with the patient's tastes. The difference between tailored and ready-made clothing might be quoted in analogy. Worse than this, such stereotyped schemes, like patent medicines, tend to defamiliarize the practitioner with the physiological principles underlying their prescription. It is relatively easy for instance to remember the caloric values of the common foodstuffs such as egg, fish, milk, meat, cheese, bread, butter and 5% vegetables. A new danger has arisen: the studious diabetic may become superior as a dietician to his doctor.

The prescription of individual diets is really quite simple. The general rule is to aim at a diet corresponding approximately to 30 to 35 calories per kilogram of body weight for an adult ambulatory person of normal configuration not engaged in heavy manual labour. The labourer needs 35 to 40 calories per kilogram of body weight. The ultimate object is to keep the patient slightly undernourished, about 10% (7 to 10 pounds) below the average weight for his height and age, according to Hutchison's tables. The obese woman of 14 stone is better off from a diabetic standpoint at 12 stone. The active man of eight stone is better kept there if his maximum weight has never exceeded nine stone. A word of warning is incidentally needed against the use of thyreoid extract to aid such weight reduction. If inanition is due to *diabetes mellitus*, the patient will gain weight on a basal diet. The next thing is to nominate the amount of protein in the diet, allowing three-quarters to one gramme per kilogram of body weight, or half a gramme only if kidney or heart disease is present. The remaining calories are apportioned between the carbohydrate and fat, so that the ketogenic : antiketogenic ratio does not exceed 1·5, that is, so that the total amount of fat equals no more than thrice the carbohydrate. The daily ration is finally divided into three meals, containing approximately equivalent amounts of carbohydrate. The older formulæ for the exact estimation of the fatty acids and glucose are little used now. Diabetic cookery is also largely out of date. Scales are of course required to weigh the food when cooked and are universally available. A good type is made by Elliott Brothers, of Sydney, provided at our suggestion with a large aluminium plate, for a cost of about seven shillings.

When this final basal or slightly suprabasal diet is attained, it is adhered to for a further ten to fourteen days, and the urine is watched as before for both sugar and ketones, while the patient's body weight is recorded twice weekly. Regard is paid to the patient's feelings, as to whether he is satisfied or eats all his food, and to his sense of well-being or otherwise under this considerably restricted *régime*. If the glycosuria disappears and the ketones remain, trial should be made of a diet wherein the fat is equivalent to twice the carbohydrate or even equal to it, if the result of the Rothera test still remains positive. Even for patients apparently stabilized on this basis, minor adjustments will require to be made from time to time and substitution tables provided. After a time, when the patient has satisfactorily passed a weight judging test under the eye of the medical attendant, food scales may be discarded.

(*c*) When such a weighed diet fails to control the glycosuria and/or ketosis, or the patient initially presents signs of marked inanition or any indications of precoma, resort to insulin is required forthwith. Confinement to bed or hospital is generally unnecessary, but prior to any use of insulin, no little time must be spent in educating each patient to every detail of insulin life. He must receive instruction in the meticulous measurement of dosage and in this regard it often helps to have the syringe re-marked in units or to present the patient with a drawing of his syringe with his dosage shown plainly upon it. Syringe and needles are best kept in a wide-mouthed jar filled with spirit. The technique of sterilization of the top of the insulin phial and of the skin are taught, and a chart is given the patient showing the serial sites for injection into arms, thighs, legs, breasts and abdomen, so that no one point is injected more often than once a fortnight. The necessity for having food and insulin together, or not at all, must be emphasized and the signs and symptoms of hypoglycæmia clearly explained, not only to the patient, but to his domestic associates. A card should be carried or sewn inside the coat, stating that the bearer is a diabetic, taking X calories daily and Y units of insulin, together with the telephone number and address of the patient and his doctor, with instructions to helpers to dissolve in water and administer the tablets of sugar in pocket or handbag.

A diet given with insulin is capable of considerable elasticity as regards its constituents, providing the total caloric value is kept a constant. This value should not in general exceed 30 to 40 calories per kilogram of body weight, for an average adult following a sedentary occupation. The ketogenic : antiketogenic ratio should not exceed unity, that is, fat should be more than twice the carbohydrate and may even equal it with advantage. High carbohydrate diets with insulin (daily carbohydrate, 100 to 300 grammes) have come to stay, as they approach more nearly a normal diet, the last remains of ketosis are vanquished, the patient looks and feels better and, paradoxically, a reduction is often permitted in the insulin dosage. The whole key lies in the correct balance between the fat and carbohydrate. This type of diet is beneficial in heart and kidney disease and in pulmonary tuberculosis. The average diet taken by a diabetic under the care of an American physician today contains 150 grammes of carbohydrate. A definite reduction also occurs in the level of blood cholesterol and blood fat.

The inital dosage of insulin is an individual matter, but usually a safe start may be made with ten to fifteen units twice daily, increased by five units per dose every three or four days until only a trace of sugar remains in the early morning specimen of urine. Attention is directed to the patient's weight, the presence or absence of ketonuria and the patient's own version as to hunger and physical capacity. The old plan of correlating the insulin dosage with the glucose in the diet is misleading, as apparently one unit of insulin can account for from one to eight grammes of glucose. If the patient requires sixty or more units daily to keep his urine sugar free, another diligent physical examination is called for to exclude the presence of overlooked sepsis, liver disease or endocrine disorder, while further appropriate biochemical and other tests are undertaken. If all such findings are still absent, the patient's comfort may be consulted by substituting double or triple strength insulin for the standard concentration of twenty units per cubic centimetre, but the patient must be informed of the enhanced danger of a mathematical error on his part. As explained, a change to a "higher carbohydrate" diet may allow of a reduction in the daily amount of insulin. If the insulin seems to have an irregular or unexpectedly slight effect, attention should be paid to the results obtained with that particular batch of the extract on other diabetics, preferably with the aid of a blood sugar curve, but nowadays most of the well-known brands of insulin can be wholly relied upon as regards a constant potency. Once insulin has become a necessity, it should be used sufficiently freely to keep the patient's urine practically sugar free. There is scant use in altering the dosage by less than five units at a time. Remember that it is better for a patient to be taking a few more units than are absolutely essential than a few less. It may take a month or so before the optimum levels of caloric intake, fatty acid : glucose ratio, and insulin are finally determined, owing to an initial improvement in the convalescing pancreas and other disordered organs concerned in carbohydrate metabolism. The patient continues with these values until some complicating factor intervenes, which unfortunately must occur sooner or later. Any hurried attempts to reduce these established requirements are to be sternly rebuked; for it looks as if a diabetic of the usual idiopathic variety, whether child, adolescent or adult, up to the age of fifty-five or thereabouts, will require insulin in some degree until old age supervenes, and this must be made clear to him from the first. If the patient looks and feels well and is able to do his job satisfactorily, we must be well satisfied if the range is between twenty and sixty units a day and

we must refrain from tempting experiments. Care should be exercised in selecting the patients who are themselves permitted to alter their own insulin dosage, but all must be exhorted to place themselves at once under direct medical observation if an infection supervenes. The time of giving the insulin in relation to the meal is of no importance and usually two (rarely one) injections a day are sufficient. With the elevated carbohydrate diets a return to three doses a day is in some respects advisable.

The question of expense of insulin must on no account be allowed to influence the practitioner, as diabetic clinics and other machinery for free supervision and cheap insulin are now available in all large cities of the world. A diabetic clinic should be associated with any hospital of over 150 beds. The same information regarding the interchange of foods, food tables, values *et cetera*, should be available to the insulin-taking diabetic. No diabetic can know too much about his disease. Indeed, it is a moot point whether every diabetic should not have the opportunity of studying the mortality statistics from coma and be shown a patient in that condition, or purposely given a mild overdose of insulin so that he may be familiar with the resulting symptoms and the rapidity of their relief by glucose.

A field inadequately traversed by both psychiatrists and writers on diabetes is that of psychotherapy. As in hyperthyreoidism, the influence of shock, worry and anxiety in precipitating the onset or aggravating the course of the malady is common knowledge. In view of the fact that, in the majority, diabetes is a functional disorder of certain endocrines and of the related sections of the sympathetic nervous system, it is readily conceivable how the disturbance could be considerably mitigated by careful and informed psychotherapy, as part of which simple heart-to-heart talks with the family physician rank high in time and importance.

Treatment in Childhood.

The main difficulty in treating coma in early childhood is to prevent breaking of the diet, to provide sufficient protein and caloric intake for rapid growth and a ready tendency to ketosis. Insulin is almost always necessary from the beginning, and fortunately hypoglycæmic reactions are rare. The diet should contain from 60 to 90 calories and from two to three grammes of protein per kilogram of body weight during the growth period, and this amount usually satisfies the child's sweet tooth. The glucose : fatty acid ratio should not be lower than 1·5 or even 2·0, and insulin should be given gradually but freely in units up to three-quarters of the equivalent number of grammes of glucose in the daily ration. A. P. Thomson holds that 100 grammes of glucose should be the minimum per day.

Treatment in the Elderly.

It is rare for diabetes in those beyond middle age not to be controlled by a "qualitative" diet. Ketosis is uncommon or quickly disappears under such simple restriction of glucose. It is well to bear in mind the danger of cardiac or cerebral catastrophe resulting from the too rapid withdrawal of glucose from the diet or from the sudden introduction of large doses of insulin. The renal threshold is usually slightly raised, but notwithstanding it is considered that an occasional trace of sugar in the urine is of no consequence and the tissues have become inured to a raised blood sugar. If ischæmic or neuritic signs are present in the lower limbs, special care must be taken of the feet.

PART II.

Treatment of Complications.

1. *Infections.*—The commonest of the infections are influenza or coryza, which are well borne and ordinarily need cause no anxiety. They call for a minor increase in insulin (five to ten units per dose). Severer infections, for example, pneumonia, gastro-enteritis *et cetera*, require a major addition of insulin (about fifty units, or twice or thrice the usual dose) with glucose, during the toxic phase. Repeated coughs or colds should direct a close attention to the lungs. In diabetic tuberculosis especially, physical signs are far behind the appearances in the radiogram, owing to the insidious nature of onset and spread. The conduct of established pulmonary tuberculosis in diabetes is difficult. The first essential is the control of toxæmia and spread by early collapse therapy and rest, even though the diabetes remains relatively undisturbed; in fact, if cachexia supervenes, the carbohydrate tolerance tends to increase for a time. This permits the use of a fuller diet, and it is said that one should aim at mild overnutrition, about 10% above normal requirements. The higher carbohydrate diets will be of value in this field.

2. *Boils.*—Boils separately do not upset the carbohydrate balance in the well-controlled diabetic, but a number together, or a carbuncle, may prove as serious as a deep abscess caused by faulty insulin technique, which so frequently plunges the patient into coma and must be diligently sought for at such a time. Lesions in the skin are ultimately less noxious when treated conservatively.

Influenzal and staphylococcal vaccines can be given to diabetics, but in a much smaller and more gradual dosage scale than is usual. Pyogenic phenomena develop more readily in greasy skins, and daily washing with a germicidal soap is to be encouraged.

3. *Ocular Complications.*—Apart from cataract and retinitis, ocular complications are commonly sudden changes in refraction. Expense and inconvenience can be spared by delaying correction until at least two months after dietetic stabilization.

4. *Feet.*—Joslin has said that the diabetic should keep his feet as clean as his face, and especial care is required for the feet and footwear of the elderly, to prevent the formation of corns and callosities, which might tempt patients to cut them. The nails should be carefully cut "on the square" to prevent a nail growing inwards, and the feet should always be well dried and powdered after washing. Corns are best left alone altogether, and "life-saver" pads of felt should be worn around callosities, or boots built up, or a metatarsal bar applied.

5. *Peripheral Neuritis.*—Peripheral neuritis may be most distressing and resistant to treatment, but massage and dry packs of a heat guaranteed against injuring the skin, and the coal tar anodynes are sometimes the only reliable measures, when pain persists in spite of a satisfactory reduction in blood sugar.

6. *Pregnancy.*—Pregnancy is a matter the desirability of which is frequently discussed with the medical attendant. Advanced diabetics rarely become pregnant, and in general it is inadvisable that they should, as miscarriage is common and the disease often hereditary. If, however, a child is greatly desired, one can assure the mother that in the average stabilized case the child is born alive, but that she herself is undergoing a slightly added risk of coma at or after labour, and of an exacerbation in her disease. Caloric intake and insulin must often be increased for a time, but this is better guided by observations on the blood than the urinary sugar. Spontaneous hypoglycæmia may occur or an increased carbohydrate tolerance during the later months of pregnancy. The risk of a confinement resembles that of a surgical operation.

7. *Surgical Operation.*—In surgical operation gas and oxygen form the best anæsthetic, and additional insulin (thirty to fifty units) and glucose (about fifty grammes) should be given just before the procedure commences.

8. *Coma.*—There is no question that patients in coma or pre-coma are better off in a hospital with adequate biochemical facilities. In the meanwhile, if there is a doubt as to whether the coma is hypoglycæmic or hyperglycæmic in character, insulin should be administered by mouth or intravenously. The recognized treatment includes a stomach wash-out and enema, with 5% glucose (at least 50 grammes) introduced by stomach tube, subcutaneously and intravenously, accompanied by an intravenous dose of 50 to 75 units of insulin. Alkali is also given by mouth or into the veins in the form of alkaline sodium phosphate and sodium bicarbonate, one drachm of each at hourly intervals, until the urine is alkaline to litmus. Cardiac stimulants, such as caffeine, sodium benzoate, camphor or digitalin, are needed for the bad cases. Prognosis and progress can be aided by serial

estimations of the blood urea and blood sugar. The insulin and glucose are repeated in equal quantities at four-hourly intervals and in half the original doses until the urine fails to give Gerhardt's reaction, when soft farinaceous foods can usually be taken.

9. *Insulin Sensitivity.*—By insulin sensitivity is meant a local reaction, immediate or delayed, at the site of an insulin injection, or the development of frank serum sickness. One method of overcoming this is to substitute the insulin used by another brand prepared from a different animal, but this is rather expensive. A better method is to desensitize the patient by means of daily intradermal injections of the offending insulin, commencing with 0·1 cubic centimetre and doubling the dose, even if several skin areas are used, until the amount required is reached, together with the administration by mouth of calcium lactate, 2·0 grammes (thirty grains) daily.

Several sample diets are appended.

CONCLUSION.

In conclusion, the four fundamentals of treatment are the elimination of all traces of ketosis, a rough correspondence between the fat and carbohydrate, the avoidance of overweight, and the anticipation of complications. It is impossible to insist too thoroughly upon the danger of half-hearted attempts to treat diabetes, or of using elegantly advertised pancreatic preparations for oral administration, or the quaint botanical cures acclaimed by the laity. Insulin properly administered to a cooperative and educated patient can control the disease to a degree whereat the life of a modern diabetic is as tolerable and enduring as it was miserable and curtailed but twelve years ago.

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SAMPLE DIETS.

(From the Diabetic Clinic, Royal Prince Alfred Hospital.)

$\frac{\text{Fatty Acid}}{\text{Glucose}} = 0·75$. Insulin: 25 units twice daily.

Calories 1,800: Protein 70, Fat 110, Carbohydrate 122.

Breakfast.

	Protein.	Fat.	Carbohydrate.
Egg, 1	6·0	6·0	
Bacon, 45 grammes (1½ ounces)	7·5	22·5	
Bread, 30 grammes (1 ounce)	3·0		18·0
Milk, 90 grammes (3 ounces)	3·0	3·0	4·5
Butter, 7·5 grammes (¼ ounce)		6·0	
5% Vegetables, 150 grammes (5 ounces)	2·5		5·0
10% Fruit, 135 grammes (4½ ounces)			13·5
	22·0	37·5	41·0

Lunch.

	Protein.	Fat.	Carbohydrate.
Meat, 60 grammes (2 ounces)	16·0	10·0	
5% Vegetables, 90 grammes (3 ounces)	1·5		3·0
10% Vegetables, 120 grammes (4 ounces)	2·0		8·0
Potato, 45 grammes (1½ ounce)	1·5		9·0
Bread, 15 grammes (½ ounce)	1·5		9·0
Butter, 30 grammes (1 ounce)		25·0	
Milk, 60 grammes (2 ounces)	2·0	2·0	3·0
10% Fruit, 75 grammes (2½ ounces)			7·5
	24·5	37·0	39·5

Tea.

	Protein.	Fat.	Carbohydrate.
Meat, 60 grammes (2 ounces)	16·0	10·0	
5% Vegetables, 150 grammes (5 ounces)	2·5		5·0
10% Vegetables, 90 grammes (3 ounces)	1·5		6·0
Milk, 60 grammes (2 ounces)	2·0	2·0	3·0
Bread, 15 grammes (½ ounce)	1·5		9·0
Butter, 30 grammes (1 ounce)		25·0	
10% Fruit, 180 grammes (6 ounces)			18·0
	23·5	37·0	41·0

Calories 1,800: Protein 70, Fat 140, Carbohydrate 51.

$\frac{\text{Fatty Acid}}{\text{Glucose}} = 1·5$. Insulin: Nil.

Breakfast.

	Protein.	Fat.	Carbohydrate.
Egg, 1	6·0	6·0	
Bacon, 75 grammes (2½ ounces)	12·5	37·5	
Bread, 15 grammes (½ ounce)	1·5		9·0
Milk, 60 grammes (2 ounces)	2·0	2·0	3·0
5% Vegetables, 120 grammes (4 ounces)	2·0		4·0
	24·0	45·5	16·0

Lunch.

	Protein.	Fat.	Carbohydrate.
Meat, 60 grammes (2 ounces)	16·0	10·0	
5% Vegetables, 90 grammes (3 ounces)	1·5		3·0
10% Vegetables, 120 grammes (4 ounces)	2·0		8·0
Milk, 30 grammes (1 ounce)	1·0	1·0	1·5
Bread, 7·5 grammes (¼ ounce)	0·5		5·0
Butter, 45 grammes (1½ ounce)		37·5	
	21·0	48·5	17·5

Tea.

	Protein.	Fat.	Carbohydrate.
Meat, 75 grammes (2½ ounces)	20·0	12·5	
Milk, 90 grammes (3 ounces)	3·0	3·0	4·5
10% Fruit, 90 grammes (3 ounces)	0·5		9·0
Butter, 37·5 grammes (1¼ ounce)		31·0	
5% Vegetables, 120 grammes (4 ounces)	2·0		4·0
	25·5	46·5	17·5

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the B.M.A. Building, Adelaide Street, Brisbane, on May 5, 1933, DR. GIFFORD CROLL in the chair.

Perspective in the Examination, Prognosis and Treatment in Heart Disease.

DR. N. W. MARKWELL read a paper entitled: "Perspective in the Examination, Prognosis and Treatment in Heart Disease" (see page 97).

DR. L. BEDFORD ELWELL wished to express his pleasure in listening to Dr. Markwell's paper. It was a most comprehensive one, and one which he had evidently been put to great pains to present. Its very comprehensiveness made the subject of discussion an embarrassing one; so many points could be brought up in such a wide subject.

On the subject of X ray examination of the heart, Dr. Markwell had said that Lewis was very keen on this, but had rather suggested that other cardiologists were not so keen. Dr. Elwell thought that most of the British school of cardiologists were very enthusiastic about X ray examination of the heart; in the London Heart Hospital and in other clinics it was done almost as a routine. In every case of mitral stenosis it was made the rule, and in early cases particularly it might be of value in diagnosis before any presystolic murmur or thrill could be detected. In the right oblique view the projection backwards of the left auricle might lead to a diagnosis where no other factor was available.

Dr. Markwell had suggested the use of X rays in the diagnosis of aortitis. In early cases of aortitis, in which disease the earlier cases were seen the better, the X ray picture might be practically the only factor in diagnosis available, apart from a positive Wassermann reaction, and Dr. Elwell thought it was invaluable in this condition. Dr. Markwell had mentioned silicosis and auricular fibrillation following on it, and this brought out another group in which screening of the heart might be very valuable, namely, in demonstrating hypertension in the pulmonary circuit. Hypertension might result in atheroma and sclerosis of the pulmonary vessels. On the screen enlargement of the pulmonary cone to the right above the auricular projection would be seen, and also very clearly in some cases, expansion and contraction of the shadows of the pulmonary vessels at the root of the lung. These would be very valuable points in diagnosis.

Discussing the subject of acute tonic heart failure, Dr. Markwell had mentioned diphtheria, which was outstanding, but acute rheumatism might be mentioned, as it was an occasional cause of acute tonic failure, although so much more rare.

Dr. Elwell thought a debt of gratitude was owing to Dr. Markwell for bringing up so many interesting phases of heart disease in such a very thorough way.

DR. ELLIS MURPHY thanked Dr. Markwell for his presentation of an interesting subject. He had dealt in a most comprehensive way with the diagnosis of cardiac conditions. Dr. Murphy considered the charts were helpful in the scheme of diagnosis. The paper was comprehensive and, as Dr. Elwell said, it was difficult to pick out points for discussion. Dr. Murphy agreed with Dr. Markwell that now practically all cases of *angina pectoris* were associated with a definite lesion, except in cases of anæmia. Till quite recently clinicians had distinguished between angina and infarct very definitely, but now it could practically be taken that the cause was the same, though in one case there was a momentary coronary incompetence and in the other a permanent coronary occlusion.

Dr. Markwell and mentioned, and one must always remember, hyperthyreoidism, which was often a comparatively obscure cause of cardiac irregularities. Dr. Murphy agreed with all that Dr. Markwell had said, and considered that the electrocardiograph was particularly illuminating in many cases in which diagnosis was otherwise obscure.

DR. J. LOCKHART GIBSON asked Dr. Markwell why the terms "dilated heart" and "hypertrophied heart" were so often used, as if synonymous. A dilated heart was surely a weakened heart, unless its muscle was also hypertrophied. A hypertrophied heart was a heart strengthened to overcome some disability and was therefore stronger than natural. They might give an equal area of increased dulness.

He was interested to hear that Dr. Markwell considered ventricular fibrillation more important than auricular fibrillation. As long ago as 1884 he had published a note in *The Lancet*, originating a physiological and clinical explanation of the mitral diastolic heart murmur. It utilized Ludwig's experiment which demonstrated that muscular action consisted of two events, active contraction and active relaxation. In a case of mitral stenosis where the heart was not competent, a marked presystolic murmur occurred when the auricle emptied itself into the imperfectly distended ventricle. When, under treatment, the ventricular muscle became stronger, the presystolic murmur became less or disappeared, and a mitral diastolic murmur developed, because the strong ventricle, when its muscular wall actively relaxed, drew the blood forcibly into and filled its cavity immediately after it had been emptied, and with such force that a murmur was produced. The ventricle was the working chamber of the heart. The thin muscular wall of the auricle could have very little power.

DR. J. MOWBRAY THOMPSON thanked Dr. Markwell for his paper, but thought he had made Dr. Thompson's own particular task rather heavier than before. Dr. Thompson examined numbers of men seeking work in the railway and tramway departments, or men wishing to insure their lives. Was it possible to say at one examination whether the heart was perfectly healthy? At one stage one was taught that a mitral murmur conducted to the axilla was serious, then again one was taught to ignore murmurs, the myocardium being the only structure of importance. What was the ordinary examiner to do?

DR. J. V. DUHIG thought Dr. Markwell's survey much too wide for detailed discussion. He would deal with only two points.

The response to coronary block, judged by considerable *post mortem* experience, was very capricious, since one observed no macroscopic lesion in many cases of sudden death, and in others evidence of very extensive infarction which had healed, still leaving a large muscular defect, even a large fibrous aneurysm, and which had not resulted in much disability. This discrepancy demanded explanation, and Dr. Duhig did not believe that of ischæmia was adequate. X ray stereographs of injected coronary systems sometimes suggested that sudden death might be due to sudden complete right branch bundle block following obstruction of the left coronary and inadequate right-sided anastomosis.

The other point was that of acute dilatation. Dr. Duhig had seen two cases *post mortem* of quadrate hearts, greatly dilated on both sides without obvious coronary disease or any lesion to account for death beyond the dilatation. He would like Dr. Markwell to suggest some cause for the conditions found.

Dr. Markwell thanked the speakers who had contributed to the discussion. He agreed with Dr. Elwell in a lot he had said. Dr. Markwell said he might have spoken more of X rays, but he had taken that line deliberately. The general practitioner or even the specialist could not submit every patient to every form of examination. X rays were used as a routine in some hospitals; it was a quick method of telling the size of the heart. Dr. Markwell thought that the size of the heart could be estimated as a rule by an ordinary clinical examination, if this were carried out properly; palpation and percussion were not enough. One must check the results from these methods by the stethoscope, using a bell chest-piece. In very fat people or in patients with marked œdema or in fibrosis of the lung or other condition which might displace the heart, X rays might be essential to tell the size of the heart. It was all very well for hospitals and some practitioners to use X rays as a routine, but the majority of practitioners could not do this; fortunately it was not necessary in the vast majority of cases, and this was the point Dr. Markwell was trying to stress in the paper. With X rays one might pick up cases that would otherwise be missed, but this might be said of any clinical method which was not used as a routine. It was also a question of probabilities. If early aortitis were present the patient would not go to a doctor unless he had symptoms, and in practice one had to work back from these symptoms, and it would be found that the patient had other signs or symptoms as well as the aortitis. One point Dr. Markwell wanted to emphasize was that the British school for the most part used the electrocardiograph in the diagnosis of myocardial involvement, and many members of that school, such as a Queenslander, Strickland Goodall, quite early recognized

its value in connexion with the myocardium. Dr. Markwell said if he had the choice of only two methods with which to examine a possible heart case, he would unhesitatingly decide on the history and the electrocardiograph and would not consider X ray examination at all. Twenty years previously X rays had been used by Vacquez and his school, and yet Vacquez, at the International Congress in London in 1913, agreed that X rays gave only an outline of the heart and gave no information as to the state of the muscle. Every heart that was damaged was not enlarged. With reference to Dr. Elwell's remarks concerning heart morbidity from infectious disease, Dr. Markwell said he was referring more to mortality in the paper. Patients did at times die from rheumatic infection, but not commonly. The great majority of hearts that suffered from acute myocarditis in acute rheumatism recovered from the attack and the heart muscle went on in the most extraordinary manner to hypertrophy to overcome the ensuing valve lesion. He had seen a heart *post mortem* at Westmoreland Street with mitral stenosis through which a pencil would hardly pass. That heart must have carried on for some time like that.

Dr. Markwell thanked Dr. Ellis Murphy for his remarks. Clinicians must differentiate between syphilis, aortic regurgitation and coronary sclerosis in cases of angina. In syphilis the condition might not be always due to a narrowed coronary vessel or vessel mouth, but in such cases he thought there might be possibly a syphilitic myocarditis, although Allbutt's theory of angina might hold, especially as it was now known that the coronary arteries supplied the first inch or so of the aorta. Cases did occur of aortic regurgitation in which there was no block in the coronary artery, but angina was present. The theory was that the low diastolic pressure in these cases caused insufficient nourishment of the heart muscle, so that pain resulted when some other circumstance, like over-exertion, became added.

Dr. Markwell replied to Dr. Gibson that the terms dilatation and hypertrophy were not used synonymously. In hypertensive disease the large heart was due to both, especially by the time the patient died. A dilated heart was not necessarily a weak heart, although it generally was. Starling, in 1915, in his "Law of the Heart", showed that dilatation was a physiological mechanism for the purpose of securing greater systolic contraction and output. Dilatation was a physiological phenomenon and, as Dr. Markwell had pointed out before, like some other useful bodily mechanisms, could get out of hand. An example of this was abscess formation which fundamentally was an attempt by Nature to localize infection and destroy germs. But this might be overdone, as in a burst appendix. Dilatation might also become so excessive that the weakened heart could not finally accede to the extra demands it made.

In reply to Dr. Duhig, Dr. Markwell said he did not know what would be the cause of death in the cases quoted, but it might have been syphilis, but if there were no hypertrophy, but purely dilatation, dilatation might have had the same mechanism as he had just described. It might have been that the heart, dilating to overcome some stress, such as syphilitic myocarditis, being unable to dilate any further, stopped, as in the experiments done by Starling, in which he slit the pericardium so that the heart could dilate further and begin to work again. Dr. Markwell thought that probably the dilatation in the heart mentioned by Dr. Duhig was comparable to this. In cases of angina one must think of aortic regurgitation, syphilitic aortitis and myocarditis, and coronary sclerosis. With regard to hypertrophy and dilatation, hypertrophy probably occurred alone originally, but by the time the heart became unhealthy and had gone through the condition of dying and had finally reached the *post mortem* table, it was very different. Dr. Markwell cited the case of an iceman who ran at least eight miles daily for nine months of the year. He was now between fifty and sixty years of age, and had been following that routine for about thirty-five years. The heart was not unduly enlarged, as shown by X ray examination made for purely academic reasons (the man was not ill), and the blood pressure was normal. The apex was, however, in the sixth interspace, the lungs having become enormously enlarged, and there was lung resonance right down to the lowest edge of the thorax. The heart was thus pushed down with the diaphragm. The heart must have been hypertrophied, but there was not much dilatation, if any.

Dr. Markwell said that he did not agree with the theory mentioned by Dr. Duhig, that sudden death in coronary obstruction of the left distending coronary artery was due to this branch supplying the right bundle by interruption of the nervous impulse. This woke up the old, now dormant neurogenic and myogenic argument.

In reply to Dr. Thompson, Dr. Markwell said it was difficult and often impossible to say whether a heart was normal by ordinary examination. Dr. Thompson's use of exercise tests was perhaps as useful as any for the purpose of the routine examination of his railway men, a history in such cases being unsatisfactory and an electrocardiogram out of the question.

NOMINATIONS AND ELECTIONS.

THE undermentioned has been nominated for election as a member of the New South Wales Branch of the British Medical Association:

Ping, Aubrey Moore, M.B., 1929 (Univ. Sydney), 43, Hall Street, Bondi.

The undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Beattie, Herbert Ronald Herschel, M.B., B.S., 1932 (Univ. Sydney), Coast Hospital, Little Bay.

Rock, Harry Owen, M.B., 1926 (Univ. Sydney), Culcairn.

Correspondence.

AN ECONOMIC COMMENTARY AND DIAGNOSIS.

SIR: "M.D., Ch.M.", in his letter of the third instant, appears to doubt my statements that Major Douglas had made an attempt to give mathematical proof of his A + B theorem and that this "proof" had been pronounced incorrect by a trained mathematician to whom I had submitted it.

In "Social Credit", 1924, by Major Douglas, this mathematical formula was stated by the author and it was to this mathematical "proof" that I referred in my letter. No amount of quibbling over the meaning of my words by my critic can alter this fact.

I would advise "M.D., Ch.M." to submit this so-called proof to any trained mathematician, for I feel sure he will be informed that it is incorrect.

This attempt to give mathematical precision to his A + B theorem discloses that Major Douglas, instead of being a "genius", as some of his followers claim, only exposes his ignorance of the science he attempts to employ to bolster up his economic reasoning.

I would also advise "M.D., Ch.M." to read my letter more carefully. He would not then make the mistake of writing that "Dr. Dane states that the controllers of three businesses have told him that they dispense purchasing power equal to the price of the goods which they produce".

My statement was that a full examination of the accountancy statistics of three large industrial concerns, as supplied to the Government statistician, disclosed that each industry in itself, or the three combined, distribute exactly enough purchasing power to buy back the whole product of each industry or the three combined, as the case may be.

These figures are incontrovertible and are a refutation of the A + B theorem.

But this theorem carries its own refutation. It commences, *vide* "M.D., Ch.M.'s" letter: "In any manufac-

turing industry." It thus claims only to explain phenomena in a limited application, that is, manufacturing or secondary industries. It does not claim, for it cannot claim, to represent phenomena in primary production.

Its applicability being thus limited, it cannot claim validity for phenomena that are universal, economically. It is thus worthless.

The followers of Major Douglas are people who, although sincere in their efforts to find a solution of our troubles, are untrained in the fundamentals of economics.

They thus delight in vague categorical and foolish dogmatic statements, such as "M.D., Ch.M." employs: "Unemployment can never again be cured by employment", "that a moment's consideration will show that this theorem is self-evident and quite unarguable."

With regard to the former dictum, it would be difficult to imagine a more foolish or meaningless statement.

Employment in its final essence is only the use of "land" by "labour". Anything which interferes with this combination is likely to lead to unemployment.

The chief of these means of interference is the monopoly toll that the private appropriation of rent permits. Further means are the robbery of the labourer of his product by taxation, and especially that most pernicious form of taxation, protective tariffs, with their further consequent increase in price.

It is apparent that unemployment can only be finally relieved by the abolition of all obstacles in the way of labour being applied to land. Money has nothing to do with the matter in the final analysis.

The latter statement is also foolish, as a considerable number of very able men have given a very great deal of consideration to the A + B theorem and have concluded that it is not a correct explanation of current phenomenon.

"M.D., Ch.M." cannot hope to convince thinking people that the whole of the personnel of the three bodies which have investigated Douglas's proposals was part of the spider-web of Wall Street. It is this wild kind of talk that passes for reason with unthinking people but carries its own condemnation to those who give deep consideration to economic problems. "M.D., Ch.M." states that the trouble is deficiency of purchasing power. With that I agree, but I cannot agree that to issue more money would cure that.

Purchasing power is not money; it is wealth produced or services rendered. The deficiency is brought about by the fact mentioned above, that the producer is robbed of part of his product by those privileged to do so, government being the chief offender in many ways. To issue more money would not end the robbery and to endeavour to control prices is futile.

I would like to inform "M.D., Ch.M." that Henry George wrote with a full knowledge of the money question, for proposals identical with the Douglassian theory had been made in England before George wrote and before "credit was so deeply centralized". Ignorant people one hundred years ago attributed their poverty to lack of money, but William Pitt, England's greatest Prime Minister, knew better than that as to the cause of their distress.

Finally, "M.D., Ch.M." suggests that a monopoly of the means of production (land) is, of course, important, but a monopoly of the means of distribution (money) is "more easily acquired and is equally efficacious in reducing the people of the world to subjection".

Now, if I could be given the monopoly of the land of Australia (the means of production), I would not in the least object to "M.D., Ch.M." having all the money, for I would simply demand it all from him for permission to live here and he would find that it was no spider-web I would ensnare him in but the iron chains of the law, both legal and economic.

How absurd it therefore is to claim that money is the supreme power.

A knowledge of psycho-analysis is not without its usefulness in economic argument. It at least discloses important reasons other than economic which induce men to wax so abstractly grandiloquent on "money" and why men, once the money complex has been aroused, stick to it with such pertinacity.

A perusal of "M.D., Ch.M.'s" paragraph upon money, Moloch lions and spider-webs of Wall Street surely indicates that his beliefs are motivated by emotional trends rather than by the dictates of reason.

Yours, etc.,

PAUL G. DANE.

110, Collins Street,
Melbourne,
June 20, 1933.

CHRONIC HYPOGLYCÆMIA.

SIR: In your issue of June 10, Dr. Ewen Downie proffers certain criticisms of our paper on chronic hypoglycæmia, published on February 18, 1933.

For his information we wish to state that the method of blood sugar estimation employed was the micromethod of Hegedorn and Jensen. Capillary blood, which is equivalent to arterial blood, is used in all micromethods, and as is well known, the sugar level in arterial blood is higher than in venous blood.

Actual estimations of the blood sugar level during acute hypoglycæmic attacks are almost invariably an impossibility, since the patient comes for treatment after and not during an attack, but surely in all diseases diagnosis is based, in a large part, on the history and symptomatology, with biochemical examinations as supporting evidence.

As it is an established fact that undue exertion in a normal individual may produce hypoglycæmic symptoms, how much more prone is the individual with a low blood sugar to such attacks?

Dr. Downie criticizes the nomenclature as being based on a small number of personally observed cases. This assumption is entirely incorrect, as the classification is based on a study of case reports together with a survey of the symptomatology in all the available literature. As a matter of fact, the number of cases seen since the paper was written has greatly increased without any necessity for altering the list.

In the present state of knowledge with the impossibility of estimating the amount of insulin in the blood, and the absence of any reliable liver function test, it is obvious that classification must be made on purely symptomatic grounds. This was pointed out in our paper.

Further observations have demonstrated the fact that cases listed under the heading of simple relative hypoglycæmia are in reality suffering from ketosis, the symptoms of which are in many cases very similar to those of chronic hypoglycæmia, and are in all respects closely allied to this entity.

A high fat-high protein diet which is still given to diabetics by many people, undoubtedly produces or exaggerates an existing ketosis, and so is ill advised in hypoglycæmia, when a ketosis so frequently occurs.

Concerning the question of the influence of lactation on blood sugar, Dr. Downie is referred to the authorities mentioned in the text of our article.

Glucose, of course, is not given merely to raise the blood sugar, but acts possibly by dehydrating the protein molecule, and certainly by exerting beneficial effects on the bowels.

We much regret that exigencies of space did not permit the inclusion of a fuller history of the twenty-five cases under review. The diagnosis of hypoglycæmia was in no case made on a single blood sugar estimation, but after careful consideration of other clinical data. In this respect the criticism of Case III is distinctly unfortunate. The prior history was undoubtedly characteristic of hypoglycæmia, subsequent blood analysis has confirmed the diagnosis which has been further strengthened by the result of treatment. Recently during a visit to the country the patient of her own volition left off glucose for a month, and suffered a return of her symptoms. The exhibition of glucose was followed by such a rapid return to health that she is herself now convinced of what previously she doubted—to wit, the hypoglycæmia hypothesis. It is almost certain that had the true condition of the particular patient been recognized earlier, there would

have been saved literally years of expensive and wearisome hospitalization.

Such recitals may seem unconvincing at a distance, but results of therapy seen at first hand in actual practice leave little room for any other opinion than that expressed in our paper. We do not blame Dr. Downie for a certain scepticism, since he may not have realized that in the comparatively cool climate of Victoria, search will be made in vain for such a high percentage of cases or varied symptomatology as found in the tropics and subtropics. We would add that far from attempting in a wave of enthusiasm to erase scientific landmarks in carbohydrate metabolism, we have merely applied well-known methods of investigation in an environment of greater heat and humidity than that which is habitual to most other white communities.

Further work which will be published later has more firmly established the existence of the hypoglycæmic entity, and leads us to believe that, as already stated, climate is of supreme importance in the production of symptoms.

Yours, etc.,

CLIVE SIPPE.
JOHN BOSTOCK.

Brisbane Clinic,
Wickham Terrace,
Brisbane,
June 22, 1933.

THE GODS OF MEDICINE.

SIR: Might I make reference to your courageous leading article which appeared in last week's issue. You omitted to mention that we were caught in a net of our own making. Our policy as a profession has always been to avoid criticism of ourselves lest we destroy the confidence of our patients. Few have doubted the soundness of this policy. Why should we be different from other sections of the community? Why not criticize ourselves in the light of pure reason? The outstanding weakness of the human race today is the failure to face up to reality. It is a world of subterfuge and illusion. The writings that appear in our journals are mainly the product of our illusionists whose exaggerated antics in the sideshows of the journals are intended to justify their failure to enter the arena of honest service to humanity.

There are many in our midst who for a lifetime have hacked their way through the forest of medical practice. And having reached the mountain top they rest awhile to survey the panorama of life and the relation of their profession to life and reality. It is then that they realize that this system of medical practice developed and attained its noblest sphere of usefulness in an agricultural era. It was the era of the family physician, family philosopher and guide, a man of broad vision and lofty ideals. With increasing industrialization began the degeneracy of the human race and coincidently the degeneracy of medical art. Now began the worship of the Goddess of Power and Wealth, of individualistic striving, of single-track mindedness. Our schools are producing an abnormal product of an abnormal era. His medical apprenticeship throws him into contact with professorial, specialistic and tutorial cliques, men who have never realized the survival value of simple cooperation in the human group, who with a fear-engendered superiority complex preach from the artificial pedestal which they create in their own minds. Knowing few other contacts, the student becomes even as they. The ambition-crazed stay in the city, they become our specialists and teachers and so the vicious cycle continues. The less ambitious journey into the agricultural areas pathologically bred. The simple country folk have inherited simple instincts related to health preservation: there follows an instinctive lack of confidence in the teaching of the degenerate city product.

The present-day graduate is merely a salvager of mankind. He must learn to be a pilot. There are many in our midst who could pilot our profession away from the destruction that threatens it on every side. Only rarely do they write in our journals. They are weary from the struggle and are afraid their opinions would be regarded as fearsome and revolutionary. They are: but the truth is necessary for our own reconstruction and the reconstruction of the health of the community. Let us hear from them.

Yours, etc.,

E. E. SMITHERS,
L.R.C.P. and S. (Edinburgh),
L.R.F.P. and S. (Glasgow).

193, Macquarie Street,
Sydney,
June 23, 1933.

DISSECTION OR DIATHERMY?

SIR: Doubtless the controversy concerning the best procedure for removal of adult tonsils will continue to rage while "the majority of those taking part in it write as champions of a pet cause, not as scientists seeking only the truth".

Dr. E. Payten Dark (THE MEDICAL JOURNAL OF AUSTRALIA, June 24, 1933) has entirely ignored what is undoubtedly the method of election with many overseas surgeons, namely, dissection under local anæsthesia.

Your contributor succinctly summarizes the advantages and disadvantages accruing to the rival methods of removal by diathermy and by dissection under general anæsthesia. When the sequelæ of dissection under local anæsthesia are considered all of these disadvantages are avoided.

During the past seven years I have practised dissection by this method and more than 250 patients have been submitted to the operation. Hospitalization, while desirable, is not essential.

Without exception patients have returned to duty before the eighth day. In no case has a general anæsthetic been found necessary to complete the operation or to deal with sequelæ. I have yet to hear the first complaint from a patient because of pain at the time of operation. When instructions are followed there are no complaints during the period of convalescence.

The risks of hæmorrhage by this method are minimal, in only 10% of cases have ligatures been required at the time of operation, and not more than five patients in the series have experienced reactionary or secondary hæmorrhage, despite the fact that most patients are allowed out of bed on the third day. Many patients have resumed duty on that day when such has not entailed frequent use of the voice.

Without exception the reactionary hæmorrhages have occurred in persons who have been subjected to the guillotine or diathermy, and whose tonsillar remnants were involved in dense adhesions to the pharyngeal constrictors. Furthermore, hæmorrhage is by no means unknown following the application of diathermy. The method has advantages apart from those consequent upon the avoidance of general anæsthesia.

The infiltration of the whole tonsillar fossa, virtual "floating" of the tonsil, facilitates dissection. Even in the presence of adhesions dissection is made easier. The method may be used with safety and indeed is advocated by some surgeons in the early stages of acute parenchymatous tonsillitis and in quinsy, provided the tonsillar projection is not sufficient to obstruct respiration when local infiltration has been added.

The only disadvantage evident is the apprehension of the patient, but as in all procedures under local anæsthesia, this "psychic shock" may be minimized by appropriate premedication.

Yours, etc.,

GEORGE A. HARDWICKE.

"Enmore House,"
Enmore,
June 24, 1933.

END RESULTS OF TONSILLECTOMY.

SIR: Dr. R. H. Bettington has given us a useful paper on the much discussed subject of tonsils and adenoids. The tonsil undergoes involution as maturity is reached,

so does the thymus, for which no definite function has been assigned; this may be merely because the body is up to then slowly reaching its fully developed state and throwing off what is not wanted. The researches of some embryologists lead to the conclusion that tonsils and adenoids are functionless. B. F. Kingsbury[1] says that the problem of their function turns a good deal on what is the function of the lymphocyte, and he goes as far as to say that the latter has no function. Writing on the development of the pharyngeal tonsil in the cat, he puts it that lymphocytes are laid down there in close association with the epithelium of the pharynx owing to certain tensions created there during early intrauterine growth. The same writer and W. M. Rogers,[2] in a paper on the development of the tonsil of the calf, state that it is interpreted as due to the peculiar growth conditions of the region, that here is a meeting point of several developing structures and some lymphoid tissue is heaped up. It may be of interest to note that the mouse, rat and guinea-pig have neither pharyngeal nor palatine tonsil.

If we are to regard tonsils and adenoids like the vermiform appendix as developmental residues in process of disappearing during the course of evolution that man is now going through, and if in a child because of chronic inflammation or abnormal hypertrophy they are causing a disability, then away with them without any compunction. It is more important to have a healthy respiratory tract. Their disease would appear to be a result of civilization, the massing of people in cities. Surgeons and anæsthetists have not been unenterprising in devising a technique and instruments to do the job well. But let us not remove tonsils without definite reasons.

Selkirk and Mitchell,[3] speaking of the indications for and results of removal of tonsils and adenoids, say: "Many of the symptoms and conditions popularly supposed to be associated ætiologically with diseased tonsils are those in which the natural course and incidence, regardless of the effect of tonsillectomy, are not known." The term "mouth-breather" is loosely used, it is hardly correct to describe a child with an habitually open mouth as a mouth-breather until he has been examined to see if he is breathing through his nose, mouth or both. Those interested should read the recent paper by James and Hastings[4] in the *Proceedings of the Royal Society of Medicine*. As to adenoids they state:

> Contrary to the generally accepted view, we are of opinion that nasal obstruction is rarely caused by adenoids. Lymphoid tissue is present in the pharynx of most children. In very few of the cases is the airway in any part of the nose or nasopharynx less than through the open glottis. The open mouth continues after the operation.

May not flat chests be due to a narrowed airway below the larynx, such as pressure of large mediastinal glands or an extra big thymus?

Tonsillectomy, like every operation, has its minimum risk. Mortality figures are difficult to arrive at. Tonsil cases seem to account for rather a large proportion of sudden deaths. In the research by the English Status Lymphaticus Committee[5] it was found that in twenty-three cases of sudden death of all ages under anæsthesia, in which no definite cause of death, apart from shock, could be found, nine were for removal of tonsils, that is, 39%. Taking separately those of children's hospital age, 0 to 11 years, out of sixteen cases of sudden death seven were tonsil cases, that is, 44%. Then there are deaths from hæmorrhage, and the fortunately rarer complications, lung abscess (peculiar to the United States of America), pneumonia *et cetera*, and later disabilities from damage to the faucial pillars and soft palate, even to the dreadful mutilations with adhesion of those structures to the posterior pharyngeal wall, described recently by W. J. Denehy and A. Amies,[6] in which cases, incidentally, true mouth-breathers are produced. These latter troubles should not happen.

Yours, etc.,

E. COUPER BLACK.

Magill Road,
Tranmere,
Adelaide,
June 26, 1933.

References.

(1) B. F. Kingsbury: "The Developmental Significance of the Mammalian Pharyngeal Tonsil: Cat", *American Journal of Anatomy*, Volume L, 1932, page 201.

(2) B. F. Kingsbury and W. M. Rogers: "The Development of the Palatine Tonsil: Calf, Bos taurus", *American Journal of Anatomy*, Volume XXXIX, 1927, page 379.

(3) T. K. Selkirk and A. G. Mitchell: "Evaluation of the Results of Tonsillectomy and Adenoidectomy", *American Journal of Diseases of Children*, Volume XLII, July, 1932, page 9.

(4) W. Warwick James and Somerville Hastings: "Discussion on Mouth Breathing and Nasal Obstruction", *Proceedings of the Royal Society of Medicine*, Volume XXV, Part 2, 1932, page 1343.

(5) M. Young and H. M. Turnbull: "An Analysis of the Data Collected by the Status Lymphaticus Investigation Committee", *Journal of Pathology and Bacteriology*, Volume XXXIV, 1931, page 213.

(6) W. J. Denehy and A. Amies: "The Treatment of Adherent and Deficient Palates", THE MEDICAL JOURNAL OF AUSTRALIA, February 4, 1933, page 150.

EARLY PULMONARY TUBERCULOSIS.

SIR: Dr. Darcy Cowan, in his splendid article on early tuberculosis in the issue of June 24, makes the following statement: "Treatment by collapse therapy has no place in a discussion on the earliest stages of pulmonary tuberculosis, but, of course, it is a very valuable method in more advanced lesions, especially when unilateral and accompanied by cavitation and hæmoptysis."

Now, it is not possible to make a positive diagnosis until quite considerable pathological changes have taken place and it is surely giving compression a very poor show in the treatment if it be reserved for the more advanced cases with cavitation and hæmoptysis. Those who do so reserve it will get but a poor opinion of its results. They will discover that the pleura is almost invariably adherent in one or more places, allowing only partial compression to be obtained. The temptation arises to try and stretch these adhesions by injecting air under pressure, with its immediate dangers, shifting of the mediastinum and pleural effusion.

Experience with collapse therapy quickly teaches one to agree with Burrell in his recent book when he indicates that the case which calls loudest for compression and in which the best result can be expected with a minimum of complications is the early exudative type with more or less extensive areas of infiltration and grave constitutional signs. The pleura is free to put the lung at rest and, most important, that spill-over is prevented which is so often responsible for the subsequent invasion of the other lung.

I do not wish to indicate that the later cases with cavities should not be collapsed if possible—the results are sometimes excellent; but one must disagree with the statement that collapse therapy has no place in the earliest stages at which it is possible to make a diagnosis.

Yours, etc.,

W. J. NEWING.

55, Collins Street,
Melbourne,
June 27, 1933.

MINERS' NYSTAGMUS.

SIR: In reply to Dr. A. E. Taylor's remarks concerning the candle power of the various lamps used in the coal mines of New South Wales. The following is a list of lamps in use in the Northern Collieries together with the candle power which each type of lamp produces.

1. John Darling Mine: Davis Derby alkaline cell electric safety lamp 1·2 to 1 candle power.

2. Wallarah Colliery: Naked light pit, candle power 1 to 5, also Halwood oil safety lamp 4 candle power.

3. Stockton Borehole Colliery: Oldham electric safety hand lamp 2 to 3 candle power, also Clapham-Johnson-Morris oil safety lamp 1 candle power.

4. Pelaw Main and Richmond Main: Oldham electric cap lamp 4 candle power. Kingsway No. 2 electric hand lamp without horizontal pillars gives 1 candle power at commencement of shift and 0·95 candle power throughout, also Clapham-Johnson-Morris oil safety lamp 1 candle power.

5. Abermain No. 1 and No. 2 and Hebburn Collieries: Nife electric safety lamp, alkaline battery, giving 1 candle power. Ceag electric safety hand lamp, acid battery, giving 1 candle power. Edison electric cap lamp with reflector giving 6 to 8 candle power, also Davis Derby oil safety lamp giving 0·75 candle power. Also electric hand and cap lamp Oldham type without reflector giving 0·83 candle power, maximum 0·96 candle power, minimum 0·71 candle power.

The difficulty which lamp manufacturers experience is in producing a lamp which will give a uniform candle power and at the same time be suitable for the miners to carry without undue inconvenience. Weight is the chief factor and so far it has been impossible both in England and elsewhere to produce a miner's lamp of uniformly high candle power yet light enough to be carried about by the men whilst at work. A good illustration of this is found in the Wolf electric safety lamp with gas filled bulb giving a standard six candle power with reflector which weighs fourteen pounds.

Dr. Taylor will find many references to this subject in the *Colliery Guardian*, and in the April 21, 1933, issue, pages 734 to 736, he will find the candle power of the various lamps compared. This applies to the English and Welsh coal fields. Some of the oil safety lamps are only 0·30 candle power and the electric safety lamps as a rule vary from 1·6 to 4·5 candle power. The Davis Haycock oil safety lamp with reflector gives 3·95 candle power. The average maximum horizontal candle power of most alkaline lamps is 4·4 candle power at the commencement of the shift and 2·03 candle power at the end of the shift. The Oldham electric cap lamps, 4 volt lead acid lamps, give 3·1 candle power at start of shift and 2·2 candle power after eight hours in use. The electric cap lamp to my mind is the most suitable type and I understand the manufacturers are experimenting at the present time with an electric cap lamp of much greater candle power than is being used now. The naked light pit lamps vary from 1 to 5 candle power according to the length of the wick used. The various types of lamps in the enclosed list are not used in New South Wales alone but are similar to those in use in some of the coal mines in England and Wales.

Dr. Taylor has expressed surprise regarding the low candle power of the lamps in use and is somewhat dubious about my facts. I am indebted to Dr. May Harris and to the management of the various mines mentioned for the assistance which they have given me in securing the necessary information regarding the lamps used. I am sure Dr. Taylor will accept this as a fact and not a fallacy

Yours, etc.,

HUGH G. ALLEN.

Union Bank Chambers,
Newcastle,
June 27, 1933

OCCIPITO-POSTERIOR PRESENTATIONS.

SIR: For a long period I have had the impression that the frequency of spontaneous rotation of occipito-posterior positions to the *symphysis pubis*, during labour, has been exaggerated.

I hope shortly to publish information in support of this attitude.

Yours, etc.,

ROLAND BEARD.

188, North Terrace,
Adelaide,
Undated.

Proceedings of the Australian Medical Boards.

TASMANIA.

THE undermentioned has been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as a duly qualified medical practitioner:

Looney, Frank Harold, M.B., B.S., 1907 (Univ. Melbourne).

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1912 *and* 1915, of New South Wales, as duly qualified medical practitioners:

Blatchley, Donald, M.B., Ch.B., 1928 (Edinburgh), Rea House, Darlinghurst.

Rex, Kenneth Edward, M.B., B.S., 1930 (Univ. Melbourne), South Yarra, Victoria.

North, Charles Everard, M.B., Ch.B., 1924 (Univ. New Zealand), Lidcombe State Hospital.

Sowter, Reginald Guy, M.R.C.S. (England), 1930, L.R.C.P. (London), 1930, 201, Macquarie Street, Sydney.

Obituary.

CHARLES ROBERT LEASE.

WE regret to announce the death of Dr. Charles Robert Lease, which occurred on July 6, 1933, at Melbourne, Victoria.

MICHAEL CARR.

WE regret to announce the death of Dr. Michael Carr, which occurred on July 7, 1933, at East Malvern, Victoria.

Corrigendum.

AN error has occurred in the article by Dr. Darcy R. W. Cowan, on pulmonary tuberculosis, which appeared in the issue of June 24. At page 767 Sir Robert Phillip is reported to have said: "I am firmly convinced that the general use of tuberculin as a diagnostic agent in childhood is not desirable." This sentence should read: "I am firmly convinced that the general use of tuberculin as a diagnostic agent in childhood is most desirable."

Books Received.

THE HOME OF MANKIND: THE STORY OF THE WORLD WE LIVE IN, by H. W. Van Loon; 1933. London: George G. Harrap and Company, Limited. Royal 8vo., pp. 506, with illustrations drawn by the author. Price: 15s. net.

FOOD AND THE PRINCIPLES OF DIETETICS, by Robert Hutchison, M.D., F.R.C.P., and V. H. Mottram, M.A.; Seventh Edition; 1933. London: Edward Arnold and Company. Demy 8vo., pp. 646, with coloured plates. Price: 21s. net.

ESSENTIALS OF MEDICAL ELECTRICITY, by Elkin Cumberbatch, M.A., B.M., D.M.R.E., M.R.C.P.; Seventh Edition, revised and enlarged; 1933. London: Henry Kimpton. Demy 8vo., pp. 522, with 15 plates and 132 illustrations. Price: 10s. 6d. net.

DEMONSTRATIONS OF PHYSICAL SIGNS IN CLINICAL SURGERY, by Hamilton Bailey, F.R.C.S.; Fourth Edition, revised and enlarged; 1933. Bristol: John Wright and Sons, Limited. Royal 8vo., pp. 308, with 335 illustrations. Price: 21s. net.

THE MEDICAL ANNUAL, 1933, edited by Carey F. Coombs, M.D., F.R.C.P., and A. Rendle Short, M.D., B.S., B.Sc., F.R.C.S.; fifty-first year; 1933. Bristol: John Wright and Sons, Limited. Demy 8vo., pp. 743, with illustrations. Price: 20s. net.

LIGHT THERAPY, by F. H. Krusen, M.D., foreword by J. A. Kolmer, M.D., Dr.P.H., S.Sc., LL.D.; 1933. New York: Paul B. Hoeber, Incorporated. Demy 8vo., pp. 200, with 33 illustrations. Price: $3.50.

ENDOCRINE MEDICINE, by William Engelbach, M.D., F.A.C.P., B.S., M.S., D.Sc., with foreword by L. F. Barker; 1932. London: Baillière, Tindall and Cox. In three volumes *plus* an index volume. Royal 8vo.; Volume I, pp. 498,; Volume II, pp. 491; Volume III, pp. 886; index, pp. 123; with 933 illustrations. Price £10.

SÉCRÉTION INTERNE ET RÉGÉNÉRESCENCE, by N. E. Ischlondsky; 1933. Paris: G. Doin and Company. Royal 8vo., pp. 342, with illustrations. Price 90 fr.

OUTWITTING OUR NERVES, by J. A. Jackson, M.D., and H. M. Salisbury; Second Edition, revised and enlarged by J. A. Jackson; 1933. Australia: Angus and Robertson, Limited. Demy 8vo., pp. 329. Price: 7s. 6d. net.

Diary for the Month.

JULY 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JULY 26.—Victorian Branch, B.M.A.: Council.
JULY 27.—South Australian Branch, B.M.A.: Branch.
JULY 27.—New South Wales Branch, B.M.A.: Branch.
JULY 28.—Queensland Branch, B.M.A.: Council.

Medical Appointments.

Dr. A. E. Williams (B.M.A.) has been appointed Medical Officer of Health at Denmark, Western Australia.

* * *

Dr. S. V. Marshall (B.M.A.) has been appointed Medical Officer of Health at Murray, Western Australia.

* * *

Dr. H. K. Fry (B.M.A.) has been reappointed an Official Visitor to the Parkside Mental Hospital, South Australia.

* * *

Dr. H. V. D. Baret (B.M.A.) has been appointed Medical Superintendent of the Coast Hospital, New South Wales.

* * *

Dr. D. R. W. Cowan (B.M.A.) has been reappointed as Honorary Physician to the Bedford Park Sanatorium, South Australia.

* * *

Dr. H. W. Wunderly (B.M.A.) has been reappointed as Honorary Physician to the Bedford Park Sanatorium, South Australia.

* * *

Dr. W. C. T. Upton (B.M.A.) has been reappointed Honorary Dermatologist at the Mareeba Babies' Hospital, South Australia.

* * *

Dr. E. J. R. Holder (B.M.A.) has been reappointed Honorary Assistant Physician at the Mareeba Babies' Hospital, South Australia.

* * *

Dr. H. H. Harrison (B.M.A.) has been appointed Government Medical Officer at Portland, New South Wales.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", page xiv.

CHILDREN'S HOSPITAL, CARLTON, VICTORIA: Medical Officers.
LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officers.
ROYAL AUSTRALIAN AIR FORCE: Medical Officer.
YALLOURN MEDICAL AND HOSPITAL SOCIETY, YALLOURN, VICTORIA: Assistant Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Combined Friendly Societies, Clarendon and Kangarilla districts. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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